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MURRAY VALLEY ENCEPHALITIS: A SURVEY OF AVIAN SERA, 1951–1952.

By S. G. ANDERSON.

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A PREVIOUS series of papers has described the 1951 outbreak of Murray Valley encephalitis. Early in the investigation of the epidemic it became evident that the disease was intimately related to the arthropod-borne encephalitides, and birds were therefore suspected to be natural hosts of the virus. The close association of the disease with water and its wide distribution along the Murray River system lent support to the hypothesis of an avian reservoir and a mosquito vector.

From July, 1951, to November, 1951, a series of trips was made to the north and north-west of Victoria and the south-eastern corner of New South Wales, where a variety of wild and domestic birds was bled. Antibody to Murray Valley encephalitis was found to be widespread in the wild birds and animals examined; it was particularly prevalent in water birds. Neutralizing antibody also showed a heavy incidence in domestic fowl living near the river, but was rarely encountered in domestic fowl in situations a mile or more distant from large areas of natural water.

Materials and Methods.

Rabbit serum saline was prepared as a 10% dilution of heated (56° C. × thirty minutes) rabbit serum in physiological saline. This medium was always used for the preparation of dilutions of Murray Valley encephalitis virus. Beef heart infusion broth and saline were both unsuitable, as dilute virus died rapidly in these diluents. The rabbits bled for the preparation of serum saline had been found previously to carry no neutralizing antibody to Murray Valley encephalitis virus.

Standard Murray Valley Encephalitis Virus.—Five intracerebral adult mouse passages were made from second egg passage virus. An emulsion of brain from the fifth mouse passage was stored in aliquots at −70° C.

Antibiotic Solution.—The standard solution was made up freshly each week. It contained 1000 o.u. calcium penicillin and 10 milligrammes of dihydrostreptomycin per millilitre. It was added in a volume of 0·05 millilitre to the serum-virus mixture, the final volume being 0·35 millilitre.

Avian Sera.—Wild birds and animals were shot and bled from the heart immediately after death, the blood being obtained by means of a Pasteur pipette. The whole blood was stored at 4° C. until it reached the field laboratory. It was found that contraction of clot occurred rapidly with many species of wild bird; the serum was usually separated from the clot within eight hours of death of the bird. Serum was stored in the frozen state.

Domestic fowl were bled from the wing vein. Shrinkage of the clot and hæmolysis occurred much more slowly than in the case of many wild bird sera.

Detection of Antibody.—The complement fixation test has been found unsuitable for the detection of antibody in certain species of wild bird. The reverse complement fixation test as described by Rice (1948), and by Downie and Macdonald (1950), has been studied in this laboratory by Miss M. Eagle. It was found to be satisfactory for titration of avian antibody of moderately high titre, but was unreliable in the detection of low titre positive sera and was not used in the present investigation.

Antibody in avian sera has been detected most satisfactorily by an intraperitoneal baby mouse neutralization test. This technique was described in connexion with western equine encephalomyelitis by Olitsky and Harford (1938) and was found by French (1952) to be eminently applicable to Murray Valley encephalitis virus.

Baby mice five to eight days old were injected intraperitoneally with 0·03 millilitre of virus dilution. Specific deaths usually occurred between six and eight days after inoculation, but surviving mice were observed for fourteen days. The 50% lethal dose for baby mice was found to be $10^{-5.8}$ of our stock Murray Valley encephalitis virus.

For the neutralization test proper the sera were heated at 56° C. for thirty minutes, and then set out in volumes of 0·15 millilitre in Wassermann tubes. To each tube 0·15 millilitre of an appropriate dilution of stock Murray Valley encephalitis virus was added (2×10^{-7}), as was also 0·05 millilitre of the standard dilution of antibiotics. The tubes were shaken and held at 4° C. for between thirty and ninety minutes. Each mixture was injected into five or six baby mice as described above.

Non-specific deaths were discarded; and if more than one such death occurred in a group, the corresponding serum was retested. If all mice survived or if there was one specific death in a group, the serum was considered to have neutralized the dilution of virus employed. If all the mice died specifically, or if there was one survivor in a group, the serum was considered not to have neutralized the dilution of virus. A result intermediate between these two was rare, but when it occurred was recorded as a doubtful positive.

Procedure and Results.

1. *Survey of Wild Birds.*

The birds were adults taken between July and November, 1951. They were found associated with the river, swamps, and small lakes near Mildura in north-western Victoria and represented species relatively common in that district. The area sampled extended from the Hattah lakes, forty miles south of Mildura, to Lake Victoria, seventy miles west-north-west of Mildura. The birds were identified by either Mr. Norman Favaloro of Mildura or Mr. Warren Hitchcock of the National Museum, Melbourne. We wish to express our deep gratitude for the ready help given by these ornithologists.

The results of the survey are presented in Table I. The infection has evidently been heavy in a wide variety of species. Of twenty-four species of land bird tested, seven species provided at least one individual carrying antibody. Of sixteen species of water bird, twelve species carried antibody.

A less detailed survey carried out between Kerang and Barmah in north-central Victoria in November, 1951, provided comparable figures. Three individuals were positive out of eleven white ibis (*Threskiornis molucca* (Cuvier)). Two out of four straw-necked ibis (*Threskiornis spinicollis* (Jameson)), eight out of ten little pied cormorants (*Phalacrocorax melanoleucos* (Vieillot)) and two of six sharp-tailed sandpipers (*Calidris acuminata* (Horsfield)) were positive.

2. *Sera from the Immature Little Pied Cormorant.*

A more intensive survey was made of the little pied cormorant than of other species. Some individuals were taken in the immediate vicinity of Mildura and a few near Barmah, but the majority were obtained from the large rookeries which occur on Lake Victoria (New South Wales), and along the nearby section of the Murray River.

Breeding of the little pied cormorant commences in south-western New South Wales in August, and the young

Table I.

Neutralizing Antibody to Murray Valley Encephalitis in Wild Birds Taken in the Mildura Area, 1951.

Bird.	Number Tested.	Number Positive for Murray Valley Encephalitis Antibody.
Predominantly land birds:		
Mallee ringneck parrot, *Barnardius barnardi* (V. and H.)	2	0
Red-backed parrot, *Psephotus hæmatonotus* (G.)	10	3
Yellow rosella, *Platycercus elegans flaveolus* G.	1	0
White cockatoo, *Kakatoë galerita* (La.)	1	0
Crested pigeon, *Ocyphaps lophotes* Temm.	2	0
Peaceful dove, *Geopelia striata tranquilla* G.	2	0
White-plumed honeyeater, *Meliphaga penicillata* G.	1	1
Black-backed magpie, *Gymnorhina tibicen* (La.)	2	0
Spurwinged plover, *Lobibyx novæ-hollandiæ* (Stephens)	1	0
Banded plover, *Zonifer tricolor* (Vieillot)	1	0
Welcome swallow, *Hirundo tahitica neoxena* G.	6	0
Willy wagtail, *Rhipidura leucophrys* (La.)	2	1
Red-kneed dotterel, *Erythrogonys cinctus* G.	4	0
Black-fronted dotterel, *Charadrius melanops* Vieillot	2	0
Black-faced wood-swallow, *Artamus cinereus* Vieillot	1	1
White-winged chough, *Corcorax melanorhamphus* (Vieillot)	10	3
Grey shrike-thrush, *Colluricincla harmonica* (La.)	1	1
Magpie-lark, *Grallina cyanoleuca* (La.)	1	0
Noisy miner, *Myzantha melanocephala* (La.)	1	0
Little crow, *Corvus bennetti* North	1	0
Raven, *Corvus coronoides* V. and H.	3	0
Whistling eagle, *Haliastur sphenurus* (Vieillot)	2	1
Wedge-tailed eagle, *Uroaëtus audax* (La.)	1	0
Galah, *Kakatoë roseicapilla* (Vieillot)	2	0
	60	11
Predominantly water birds:		
Native hen, *Tribonyx ventralis* (G.)	4	2
Dusky moorhen, *Gallinula tenebrosa* G.	4	2
Coot, *Fulica atra* L.	16	2
Little grebe, *Podiceps novæ-hollandiæ* Stephens	1	0
Hoary-headed grebe, *P. poliocephalus* J. and S.	11	0
White-faced heron, *Notophoyx novæ-hollandiæ* (La.)	6	5
Nankeen night heron, *Nycticorax caledonicus* (Gm.)	5	3
Musk duck, *Biziura lobata* (Shaw)	7	4
Black swan, *Cygnus atratus* (La.)	13	5
Little pied cormorant, *Phalacrocorax melanoleucos* (Vieillot)	12	7
Little black cormorant, *Phalacrocorax sulcirostris* (Brandt)	4	2
Big black cormorant, *Phalacrocorax carbo* (L.)	4	4
Silver gull, *Larus novæ-hollandiæ* Stephens	3	0
White-headed stilt, *Himantopus himantopus* L.	2	0
Darter, *Anhinga rufa* (Daudin)	5	3
Wood duck, *Chenonetta jubata* (La.)	2	1
	99	40

are hatched in large numbers from September to February. During the months of September to November, 1951, both fertile eggs and young of this species were taken. It is possible to determine the age of the young birds to within a day or so up to the age of ten days, and to within a week up to the age of about six weeks. During the first year of life the young bird exhibits a plumage which distinguishes it from birds hatched in earlier years.

Table II records the presence of antibody to Murray Valley encephalitis virus in the sera of many of the immature little pied cormorants examined. This antibody was demonstrable even in birds just emerging from the egg, and we have no doubt is maternal in origin. This belief is supported by three other observations. Firstly, epidemiological evidence obtained at the end of the 1951-

1952 summer strongly indicated that there had been no Murray Valley encephalitis virus in the Mildura region during that summer (Anderson, 1953).

Secondly, six eggs from the little pied cormorant were examined, unheated yolk being substituted for serum in the neutralization test described above. All of the six specimens so tested neutralized virus. Although we cannot be certain that the neutralization was due to antibody specific to the virus, this is strongly suspected to be the case.

Thirdly, immature little pied cormorants, taken at Lake Victoria in the spring of 1951, were found not to be carrying Murray Valley encephalitis virus in the blood. For this purpose whole blood was citrated immediately it was removed from the heart of the young cormorant. A volume of 0·5 millilitre was immediately sealed in an ampoule by the use of a portable blowlamp; the sealed ampoule was stored in a vacuum flask containing solid carbon dioxide (−70° C.).

In the laboratory the frozen blood was thawed and inoculated on to the chorio-allantoic membrane of fertile eggs. The majority of the samples were examined by Mr. E. L. French; neither he nor the author was able to detect Murray Valley encephalitis virus in any one of 94 samples examined.

TABLE II.

Neutralizing Antibody to Murray Valley Encephalitis in Immature Little Pied Cormorants (Phalacrocorax melanoleucos), Northern Victoria, 1951–1952.

Age in Weeks.	Number Tested.	Number Positive.	Number Doubtful Positive.
<1	22	12	2
1–	3	1	0
2–	17	5	2
4–	12	3	1
8–15	7	4	1
26 (approx.)	6	0	0

3. *Sera of Domestic Fowl (Gallus gallus (Linné)).*

In October, 1951, sera were obtained from 56 domestic fowls near Mildura. Thirty-one of these were located in four runs all within two hundred yards of the lagoons along the Murray River. Twenty-two of the 31 carried Murray Valley encephalitis neutralizing antibody. The other 25 sera were obtained from fowls in three runs situated in the township and countryside, each being between one and two miles from the nearest large area of natural water. Only three of the 25 carried antibody. The greater concentration of infected fowls near water would again emphasize the probable concentration of the major vector of the virus near natural water.

4. *Ectoparasites.*

During the obtaining of sera, particularly from adult and immature little pied cormorants, search was made for ectoparasites, both on the birds and in their nests.

Mallophaga were plentiful on the birds; preserved specimens were kindly identified for us by Mr. C. Oke of the National Museum, Melbourne. These were not considered as likely vectors of Murray Valley encephalitis. Other types of ectoparasites were found only very rarely, certainly not in sufficient numbers to be an important vector of Murray Valley encephalitis virus.

A careful examination of cormorant nests likewise failed to detect a likely vector. Several whole nests, currently occupied by little pied cormorants approximately a month old, were examined in the field by Dr. M. J. Mackerras and Miss N. Kent. Fifteen similar nests were brought to Melbourne sealed in paper bags; they were then examined by Mr. C. Oke. The number of ectoparasites found in each instance was negligible.

Discussion.

Infection of domestic fowl with the virus of Murray Valley encephalitis is followed by a viræmia lasting several days, and later by the appearance of neutralizing antibody in the circulation (French, 1952; McLean, 1953).

We assume that the same series of events may occur in certain species of wild birds. In the present study we have accepted a neutralization index of 60 or more in the heated serum of an adult bird as evidence that the bird has previously recovered from infection with Murray Valley encephalitis virus.

The conclusion from the above observations is that many species of wild bird were infected during the epidemic of encephalitis in northern Victoria in 1951. This invites the belief that these same species, and probably others also, comprise the major natural hosts of the Murray Valley encephalitis virus. The presumed transient viræmia which follows these natural infections would provide ample opportunity for the infection of large numbers of mosquitoes during the epidemic season. We consider infected domestic fowl to be a similarly potent transient source of virus for the mosquito.

The situation is apparently closely analogous to that described for Japanese B encephalitis by Hammon, Reeves and Sather (1952). These workers reported a high proportion of positive sera in wild birds in Tokyo and Guam.

The avian serology reported in the present paper is consistent with the major vector of Murray Valley encephalitis virus being a mosquito associated with areas of natural water near a river. This, however, does not eliminate the possibility that domestic mosquitoes may be involved occasionally. We may imagine a cycle in which *Culex annulirostris* or some other river bottom mosquito becomes infected by feeding on a viræmic cormorant near the river, and transfers the virus by biting either man or a domestic fowl near the river. Several days later a member of the *Culex fatigans* group, bred in a domestic tank, may become infected from the domestic fowl, and in turn bite a human in the adjacent homestead.

The permanent reservoir of Murray Valley encephalitis virus has not been demonstrated. One may postulate this as being a blood-sucking arthropod, probably a mosquito, which harbours the virus for long periods; but it seems equally likely that in the as yet undefined endemic foci of Murray Valley encephalitis there is a slow but constant circulation of virus between bird and mosquito.

The virus of Murray Valley encephalitis is evidently not highly specific in its host requirements, and may well have a broad spectrum of suitable vectors. With the development of further knowledge it may be possible to delineate the dominant pattern of the ecology of the virus; but we suspect that there will be many minor pathways through which the virus may survive, and even occasionally infect man.

In the previous study of the geographical distribution of Murray Valley encephalitis virus, great use was made of the serology of humans and horses. With the current demonstration of the widespread infection of riverside domestic fowl we are provided with a more convenient index of infection. The use of "sentinel" fowls for the detection of the presence of an infective agent is not new, but the method seems particularly appropriate to the epidemiological study of a virus which is primarily an avian parasite.

Summary.

Wild and domestic birds in the Murray Valley, Australia, have been examined for neutralizing antibody to Murray Valley encephalitis virus.

The antibody was found in many species of wild bird, and particularly in water birds.

The majority of domestic fowl, *Gallus gallus* (Linné), living near the Murray River carried such antibody, but a lower proportion of domestic fowl living more than a mile from natural water was positive.

Maternal antibody is transmitted to the immature little pied cormorant, *Phalacrocorax melanoleucos* (Vieillot).

It is believed that many species of wild bird, and domestic fowl, are transient natural hosts of the virus.

Acknowledgements.

It is a pleasure to record the considerable help received from many friends. Among those who assisted in the collection of birds we mention Mr. C. W. Brazenor of the National Museum, Melbourne; Mr. N. J. Favaloro, of Mildura; Dr. K. B. Fraser, of Aberdeen; Dr. L. Fraser, of Aberdeen; Mr. E. L. French, of the Walter and Eliza Hall Institute; Mr. W. B. Hitchcock, of the National Museum, Melbourne; Dr. B. Marmion, of Cambridge; Dr. M. J. Mackerras, of Brisbane; Dr. D. M. McLean, of the Walter and Eliza Hall Institute; Mr. R. T. M. Prescott, of the National Museum, Melbourne; Dr. W. J. Stevenson, of the Victorian Public Health Department.

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THE MORTALITY IN AUSTRALIA FROM TYPHUS, TYPHOID FEVER AND INFECTIONS OF THE BOWEL.

By H. O. LANCASTER,

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FOLLOWING other papers on infective disease (Lancaster, 1952*a* and *b*), I shall analyse here the mortality from bowel infections. It is convenient to follow the lead of Cumpston and McCallum (1927) and to deal not only with the "enteric" infections, the dysenteries, diarrhœas and gastro-enteritides, but also with typhus fever, which could not be differentiated from typhoid in the earlier days in Australia. Although no case of Asiatic cholera occurred in Australia over the years 1908 to 1945, it is convenient to mention it here also.

The Mortality from Infective Bowel Diseases in Australia.

Cumpston and McCallum (1927) have given an excellent account of the bowel infections in Australia from the time of the First Fleet to the date of their publication. No better account perhaps can be given of the general epidemiology of these diseases in Australia than the remarks of Cumpston, then Director-General of Health, in his introduction to the book, which is quoted here by permission. Cumpston stated:

> During the first sixty years of Australian history the disease [typhoid] was endemic, sporadic, and associated, causally, in all peoples' minds with polluted water. In whatever direction settlement was pushed, there occurred typhoid fever. While settlement was scattered typhoid fever was scattered and of low incidence. As settlement became more dense in any locality, so did typhoid fever become more prevalent, and, it is important to note, more severe.
>
> This general principle became intensified on occasions of rapid and disorderly aggregations of persons in any locality. The most typical examples of this were seen in camps of gold-seekers or railway construction men. Under these circumstances typhoid fever literally "broke out", striking right and left, and increasing in killing power as it spread until in accordance with the established laws of epidemics it reached its maximum of infecting power and of killing power, and settled down to its steady endemic level. This was seen over and over again from Bendigo in 1851-4 to Kalgoorlie in 1894-5; but although the lesson was learned early, and the relation between typhoid fever and absence of ordered sanitation soon perceived, it was not properly accepted as the basis for official action until about fifty years after the knowledge was available.
>
> So also with towns, while there was no ordered sanitation there was typhoid fever; and, with the introduction of ordered sanitation, the control of typhoid fever began. As is seen later in discussing the effect of the introduction of sewerage systems into Australian towns on the incidence of intestinal infection, the improvement in the standard of sanitation in a community, other than the installation of a sewerage system, has improved the typhoid position to an important degree, but has had little effect upon the rates of mortality from the diarrhœal diseases. The installation of a sewerage system, however, has increased the rate of decline of the mortality rates from typhoid fever, and has also lowered the rates of mortality from the diarrhœal diseases.
>
> Moreover, the installation of a sewerage system has placed the whole position beyond the possibility of temporary dislocation by interference with the more primitive systems of sanitation otherwise available.
>
> It may be said that it was only with the commencement of the twentieth century that Australia as a whole developed an informed and sensitive sanitary conscience. Consequently, the history of the intestinal infections has two phases—the nineteenth century of increasing incidence, of explosive manifestations, of general fluctuations and of spasmodic but ineffectual control; the twentieth century of progressive and orderly control to a point far below that of any period of the nineteenth century. That the present level is as yet unsatisfactory may be seen by the following table, which shows that Australia, with an intelligent, educated and economically happy people, yet permits a typhoid fever death-rate which is a reproach.

Cumpston then goes on to show in the table he has mentioned that the mortality in Australia was about three times the New Zealand rate and almost four times the Scottish. He points out that there is still ample scope for improvement in these mortality rates. The tables given in this paper form a sort of epilogue to Cumpston's remarks on these diseases; typhoid and the diarrhœal diseases have practically disappeared as a cause of mortality. There is now great difficulty in obtaining suitable material for teaching purposes.

In Table I the death rates by age and sex from typhoid fever are given for the usual age groups and five periods. There tends to be a maximum for the mortality rates in the age group twenty-five to thirty-four years, in the earlier periods for either sex. In the two later periods this maximum is less evident and the disease appears to be causing the same mortality approximately at all ages. By comparison of the death rates for any age group from period to period, considerable falls are to be noted. Indeed, typhoid fever is the usual example given as a proof of the efficacy of public health measures. This is unfortunate in many ways, as for many years it turned attention away from the high mortality still being caused by other bowel infections and may have given some of the laity the impression that it perhaps was the only triumph of hygienic measures.

TABLE I.

The Death Rates in Australia from Typhoid and Paratyphoid Fevers.

Period.	Sex.	The Death Rates per Million *per Annum* at Ages (Years).									
		0 to 4	5 to 14	15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over.	All Ages.
1908 to 1910	M.	22	74	306	345	242	171	129	46	33	193
1911 to 1920	M.	19	35	176	216	144	116	78	53	28	115
1921 to 1930	M.	5	16	60	53	53	45	40	24	2	38
1931 to 1940	M.	2	4	12	19	13	12	13	15	6	11
1941 to 1945	M.	2	1	3	4	5	7	5	6	3	4
1908 to 1910	F.	28	87	201	183	133	97	51	56	13	123
1911 to 1920	F.	19	47	118	89	69	50	45	40	16	67
1921 to 1930	F.	8	22	42	31	28	29	24	12	9	26
1931 to 1940	F.	2	4	9	8	9	7	7	4	3	7
1941 to 1945	F.	4	1	2	2	4	2	2	8	2	3

TABLE II.

The Mortality from Dysenteries in Australia.

Period.	Sex.	Deaths from Dysentery per Million *per Annum* at Ages (Years).									
		0 to 4	5 to 14	15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over.	All Ages.
1908 to 1910	M.	13	3	7	12	20	33	99	219	574	31
1911 to 1920	M.	12	2	7	12	15	33	51	132	325	22
1921 to 1930	M.	62	2	2	4	10	16	26	65	177	18
1931 to 1940	M.	29	1	0	4	4	6	17	29	69	8
1941 to 1945	M.	14	1	2	1	2	7	15	32	53	7
1908 to 1910	F.	15	1	5	9	3	23	58	151	253	17
1911 to 1920	F.	9	1	2	5	8	18	30	97	299	14
1921 to 1930	F.	40	2	3	2	9	10	24	68	131	14
1931 to 1940	F.	20	1	1	2	1	6	9	33	57	7
1941 to 1945	F.	7	2	2	2	4	5	9	34	51	4

TABLE III.

The Death Rates from Gastro-enteritis in Australia.

Period.	Sex.	The Deaths from Gastro-enteritis per Million *per Annum* at Ages (Years).									
		0 to 4	5 to 14	15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over.	All Ages.
1908 to 1910	M.	6843	55	26	36	77	153	392	1495	4061	963
1911 to 1920	M.	5678	56	20	38	69	120	304	825	2915	767
1921 to 1930	M.	3195	32	13	16	36	66	149	397	1527	400
1931 to 1940	M.	910	21	10	15	24	40	69	172	537	115
1941 to 1945	M.	686	23	7	8	11	31	49	137	502	95
1908 to 1910	F.	5735	73	30	44	89	161	387	1433	4007	867
1911 to 1920	F.	4626	54	25	51	71	124	304	984	3237	669
1921 to 1930	F.	2532	27	17	24	40	74	140	436	1537	337
1931 to 1940	F.	719	19	12	20	28	47	76	177	613	104
1941 to 1945	F.	543	15	9	19	19	33	40	119	595	87

TABLE IV.

The Mortality in Childhood in Australia from Infections of the Bowel.

Period.	Sex.	The Deaths from Gastro-enteritis and Dysentery per Million *per Annum* at Ages (Years).								
		0	1	2	3	4	0 to 4	5 to 9	10 to 14	1 to 4
1908 to 1910	M.	23,879	5676	847	486	284	6856	82	32	1886
1911 to 1920	M.	18,224	4923	991	454	220	5690	87	24	1686
1921 to 1930	M.	10,783	3255	677	348	197	3257	53	15	1132
1931 to 1940	M.	2813	1081	360	191	106	937	33	11	433
1941 to 1945	M.	2076	688	235	117	81	700	40	9	290
1908 to 1910	F.	20,616	5034	1031	481	239	5750	115	21	1743
1911 to 1920	F.	14,873	4395	921	417	242	4635	82	25	1527
1921 to 1930	F.	8451	2817	576	262	157	2572	43	12	964
1931 to 1940	F.	2158	890	318	141	99	739	28	11	406
1941 to 1945	F.	1496	701	144	125	80	550	25	7	273

Some causes for the passing of typhoid fever may be given. First of all, as suggested above by Cumpston, typhoid fever in Australia has been a disease of the pioneering communities. This may explain the relative mode for the mortality at ages twenty-five to thirty-four years noted above. In the earlier days of settlement in country areas, water was often obtained from shallow wells and no hygienic arrangements for the disposal of fæces existed. However, with improved communications and greater prosperity, tanks became available in the country areas and other hygienic precautions were taken to prevent the rapid passage of bowel organisms from person to person. In towns and cities effectively pure water services were provided. Moreover, in the epoch studied here the horse was replaced by the motor-car as the principal means of transport. This greatly diminished the fly menace in the cities. It is of interest to compare the mortality in the different Australian States for a period, say 1911 to 1920, for which years the deaths have been tabulated in the report on the Census of 1911.

If the proportion of typhoid deaths to all deaths is computed for each of the States, then it is found that the typhoid deaths were relatively more numerous in the pioneering States and least in the well settled and urbanized States. Somewhat similar results may be obtained by comparing the proportion of typhoid deaths in the occupations—a rather hazardous procedure since no simultaneous break down by age is possible. Transport and communications, pastoral and mining all show high proportions, as would be expected on general grounds. Summing up, typhoid fever was an important cause of mortality at the beginning of the century in Australia but is now quite unimportant.

The Dysenteries as a Cause of Mortality in Australia.

In the official statistics of Australia the amœbic form of dysentery was not separately distinguished until 1928. For the years 1928 to 1945 the deaths from amœbic dysentery of 63 males and 15 females have been recorded, amounting to some 11% of the male dysentery deaths and 3% of the female dysentery deaths. It is found that of the deaths of males, 15 occurred in New South Wales, 19 in Victoria, six in Queensland, 14 in South Australia and eight in Western Australia. For females the corresponding numbers of deaths by State were four, three, six, two, nil. The death of one male occurred in Tasmania. The distribution of these deaths predominantly in the non-tropical States suggests that they may be the deaths of seamen rather than of the indigenous population.

Not all cases of amœbic dysentery will have been diagnosed as such and some may, of course, have been diagnosed as a liver abscess, hepatitis and the like. However, it is evident from the official statistics and from clinical experience that amœbic dysentery is not a common syndrome or cause of death in Australia, even in the tropical areas, although it was not rare in our troops in either the First or Second World Wars. A survey of soldiers in the Second World War (Lancaster, 1953) showed that although *Entamœba histolytica* could be found in a single examination in 1·4% (three out of 175) of young men who had never left Australia, yet the rate was approximately 10% in those who had had service in the Middle East. A large reservoir of carriers, therefore, exists in the Australian population. Since the amœbic deaths form such a small proportion of the total dysentery deaths, it has not seemed worth while to attempt an analysis of the dysentery deaths by causative organism.

The death rates from all dysenteries are given in Table II at all ages; the rates are higher for the males. This may be an occupational effect in the active years of life. In both sexes the death rates are high in the later years of life, suggesting that some considerable proportion of the deaths have occurred among the feeble and elderly, including probably also the institutionalized. The high incidence among the insane and elderly demented is well known.

Gastro-Enteritis.

In the years 1908 to 1945 gastro-enteritis deaths were referred to two rubrics of the *International List*, the deaths of all persons over the age of two years from gastro-enteritis being assigned to a rubric among the acute infectious diseases, and deaths of children under two years of age being assigned to a rubric in the diseases of the digestive system. The most recent revision of the *List* has removed this anomaly and all deaths are now assigned to a rubric among the infective and parasitic diseases. The deaths from gastro-enteritis and from dysentery can probably only have been distinguished with difficulty in the pre-bacteriological days and we may regard the division as arbitrary even now. The death rates from gastro-enteritis are given in Table III for the usual age groups.

The causes for the decline in mortality will not be discussed here; the tables are merely to show that there has been a great decline in the death rates from this disease. In childhood in general we find the male rates heavier than the female, but after the age of fifteen years the female rates appear to be the heavier. Examining the death rates for either sex for any period, the rates are high in infancy, decline to a minimum at ages fifteen to twenty-four years, and then rise throughout life. Examining the mortality for any age group in Tables III and IV or Figure I, over the five periods a very notable decline is apparent. Especially marked is the decline for the first year of life.

In Table IV I have given the mortality in childhood for infections of the bowel in detail by age. These infections include dysentery and gastro-enteritis. In 1908 to 1910, acute bowel infections were responsible for some 24 deaths for every thousand births of males and about 21 deaths for every thousand female births. In 1941 to 1945 these rates had declined to 2‰ and 1·5‰ respectively. Large declines also occurred among the older children.

Cholera and Typhus.

Cholera in Australia.

An account of the chief features of cholera and the possibilities of its introduction into Australia has been given by Elkington (1916), who noted that it had been introduced on one occasion in 1885. No cases occurred in Australia over the years 1908 to 1945.

Typhus as a Cause of Mortality in Australia.

Cumpston and McCallum (1927) pointed out the great difficulty in separating typhus from typhoid fever in the earlier records. Over the years of the mortality survey, 1908 to 1945, of course a clinical separation was practicable. The disease does not occur in Australia in its classical louse-borne form, but murine typhus occurs in the ports and scrub typhus and related forms occur in the tropical areas, such as in the northern Queensland coast. No deaths were reported in Australia for the years 1908 to 1920. Hone (1922) reported cases of typhus occurring in men associated with the handling of wheat in Adelaide, South Australia. Wheatland (1926) reported an epidemic in Queensland. Adey (1930) gave a detailed report on the Australian experiences. Langan and Mathew (1935) established the existence of scrub typhus in Queensland. These clinical researches have had their effect on the number of cases of typhus reported. Between 1921 and 1930 eleven males and four females were certified as dying from typhus. In the next decade, 1931 to 1940, there were 36 male and eight female deaths, while in 1941 to 1945 there were 35 male and 16 female deaths. In New South Wales, three deaths due to typhus were recorded in 1931 to 1940, nil in Victoria, one in Tasmania, five in South Australia, 13 in Western Australia and 22 in Queensland. It may be said that the few cases in the southern States are murine typhus, whereas those of Queensland and Western Australia are almost entirely scrub typhus.

Acknowledgements.

This paper is published with the permission of the Director-General of Health, Commonwealth of Australia, Dr. A. J. Metcalfe. My thanks are due to Dr. J. H. L.

Cumpston, a former Director-General of Health, from whose book (Cumpston and McCallum, 1927) I have extensively quoted.

Summary.

The mortality in Australia over the years 1908 to 1945 from infections of the bowel has been analysed. There has been a great fall in the mortality from typhoid fever over this period; it is now a quite minor cause of mortality. Gastro-enteritis and the dysenteries were formerly a great cause of mortality at all ages, especially in infancy, but are now a cause of fewer deaths. Cholera and typhus have not been important causes of death in Australia over the period of the survey.

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EFFECTS OF SHOES ON FOOT FORM: AN ANATOMICAL EXPERIMENT.

By F. W. KNOWLES,

Lecturer in Anatomy, University of Queensland.

AMONG the common ailments which afflict the foot of civilized man, many can be traced to modern footwear. The study and treatment of such ailments often do not come to the attention of the medical profession and are left to a separate profession of skilled auxiliaries, the chiropodists. The qualified chiropodist plays an important part in the treatment of these conditions, but as important ætiological factors are beyond his control, his measures are mainly palliative and are often not entirely successful.

Foot disorders due to badly designed footwear and outside the chiropodist's scope often confront the orthopædic surgeon with difficult problems. There is no doubt that the surgeon can do much to correct foot deformities and that he does prescribe special footwear; but little attention has been given to the study of the shape and construction of footwear generally, with a view to preventing the common ailments or defects which are precipitated by the wearing of conventional footwear.

The investigation here described was therefore carried out to see what light could be thrown on this important problem.

The plantar aspect of the weight-bearing foot, as seen in infants, or in adults who have not used footwear, has a substantially triangular outline (Figures I and IIA), with almost linear medial and lateral borders and an arcuate anterior border (interrupted lines, Figure I). The posterior angle is truncated by a large radius. The modern woman's shoe differs strikingly from this (Figure IIB), and its effect on the foot after many years' wear might be regarded as a partial adaptation of this part of the human organism to a constricting environment. Though some feet resist this environment more successfully than others, and the degree of constriction varies a little with the choice and fitting of shoes, on the whole the tendency is to convert the original triangular plantar aspect into a quadrangular one, mainly by breaking the medial border in the region of the metartarso-phalangeal joint of the hallux (Figure IIC). The toes, originally divergent as the rays of a fan (Figure IIIA) and with spaces between them, become convergent, overlapping, and crumpled. The reinforced toe-region of the shoe (whether the superficial toe-cap is present or not matters little) ensures the rigid compression of the toes, whilst the softer vamp yields, as indicated by the interrupted lines in Figure IIB, so that relative splaying of the metatarsus accentuates the quadrangular effect. (The little toe, even in aboriginal feet, is often excepted from the general divergence, and may be adducted or curved a little towards the other toes.)

Among unshod peoples, deformities of congenital, traumatic and other origin do, of course, occur, and so far as the hallux is concerned they may in some instances resemble deformities of the shod, but this is very uncommon. Such rarities have been the refuge of those who deny that modern conventional footwear deforms the feet and have been magnified by enthusiastic defenders of present fashions. However, among the white races, and also among coloured races living in white communities and wearing modern shoes, acquired foot deformities are the rule, and they are worse in women than in men and increase with age. Thus it is found that a substantial proportion of the aged are more or less disabled by foot troubles which mar the evening of their lives. Recent surveys of the feet of school children indicate that the children of today are heading for the same misery (Figure IIIB).

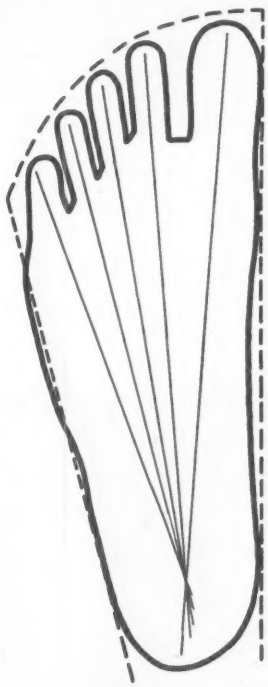

FIGURE I.

Plantar aspect of weight-bearing, undistorted foot showing substantially triangular outline.

It is customary to throw the blame on the public, to exhort them to buy "sensible" shoes, and to call for the training of shoe fitters. To those who can see the issue in all its simplicity, this is a pathetic state of affairs; no amount of measurement of feet with impressive apparatus, "scientific" training of shoe fitters, and use of X-ray equipment can compensate for the gross discrepancy between the triangular outline of the plantar aspect of the natural foot and the unrelated pointed, tapering or rounded outlines of modern shoes.

The present experiment was based on the assumption that modern shoes distort the feet, and was designed to throw light on the possible reversibility of such distortion. If fashionable shoes have deformed our feet, perhaps the wearing of "naturally" shaped shoes may permit some degree of return towards the original foot form, and thereby reverse or prevent progression of pathological effects, or at least alleviate their symptoms.

Such shoes perhaps would not at first have a universal appeal, but even among women there are large groups who would appreciate them almost immediately. Busy housewives who work in house and garden in slippers and

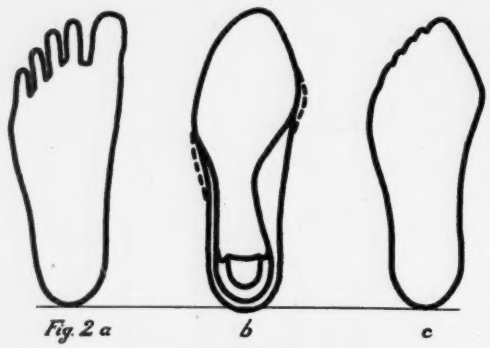

FIGURE II.
a: Plantar aspect of undistorted foot. *b*: Plantar aspect of conventional shoe. *c*: Plantar aspect of foot distorted to fit into "*b*".

other ill-shaped substitutes in an attempt to escape from the discomfort of their more elegant shoes would welcome a practical, comfortable shoe for the working day, preferring to follow the dictates of fashion only when occasion demanded. Many middle-aged men and women without

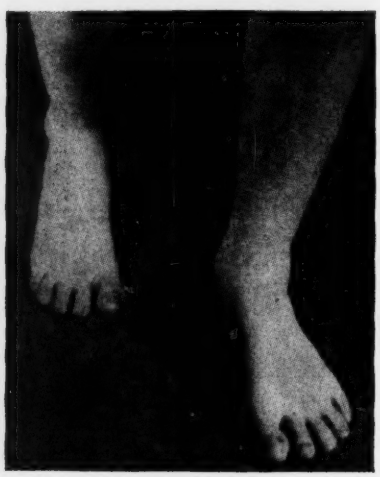

FIGURE IIIA.
Reproduced with the permission of the Director of the British Boot, Shoe and Allied Trades Research Association. Unspoilt feet are shapely and attractive but 150 school children had to be inspected before this boy was found.

doubt want only comfort and useful service from their footwear, and would ignore present fashions if such shoes were available.

Materials required for this experiment were firstly "civilized" feet and secondly shoes of a suitable size, which are not made "to measure" or to "fit" in the conventional sense but rather to the shape and proportions that would fully accommodate natural feet, such as aboriginal feet. Whilst the first of these materials was readily available, the second was not, as neither shoe manufacturers nor modern bespoke shoemakers made them. Special lasts therefore had to be constructed for this experiment.

These lasts were given a substantially triangular plantar aspect. They were not made "to measure" in the bespoke shoemaker's sense, but merely of suitable length and of

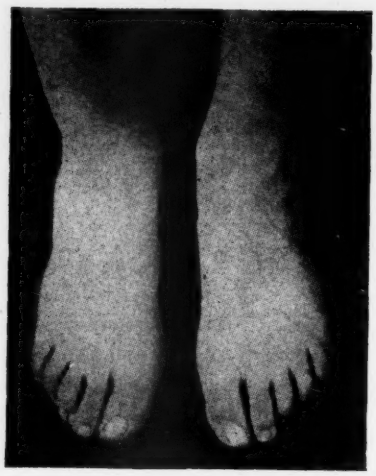

FIGURE IIIB.
Reproduced with the permission of the Director of the British Boot, Shoe and Allied Trades Research Association. This is typical of most children's feet today, but it need not be. Big toes bent over; other toes crooked and crowded and unable to do their job properly; corns on little toes.

average shape and width, in order that the results of this experiment, if favourable, might attract the interest of such shoe manufacturers as might be ready to take advantage of a demand by certain members of the public for rational footwear.

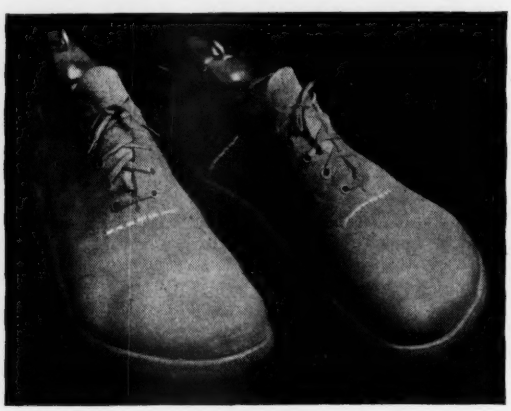

FIGURE IV.
Shoes of "natural" shape, designed specially for the experiment.

Several pairs of shoes (Figure IV) were made on these lasts and have been worn for three and a half years to the exclusion of all other boots, shoes, slippers and sandals. X-ray photographs of the feet were taken on the day of the permanent change-over in August, 1948; first in con-

ILLUSTRATIONS TO THE ARTICLE BY F. W. KNOWLES.

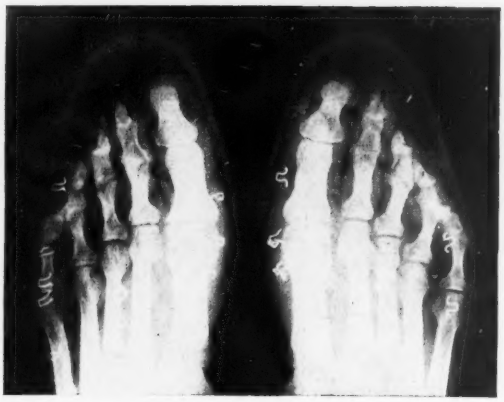

FIGURE VA.

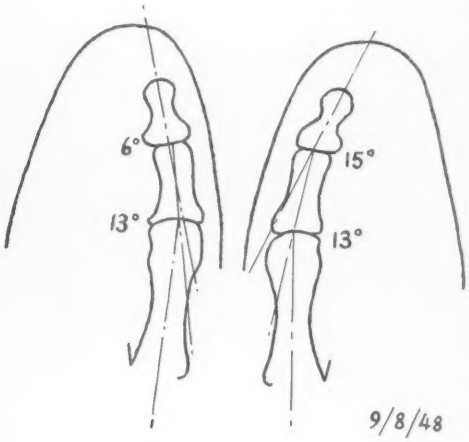

FIGURE VB.

At the beginning of the experiment, in conventional shoes.

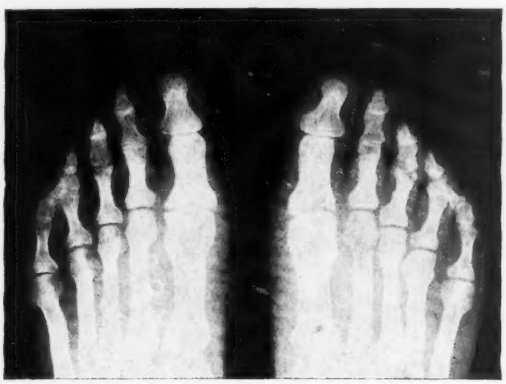

FIGURE VIA.

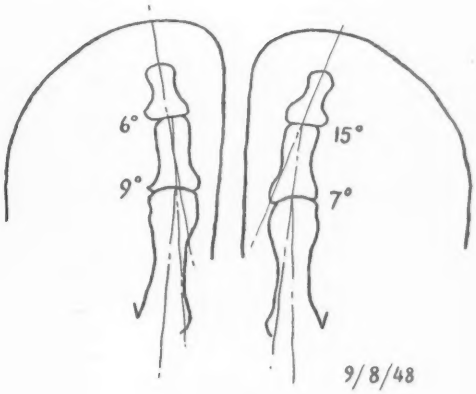

FIGURE VIB.

Also at the beginning of the experiment, but in "experimental" shoes.

FIGURE VIIA.

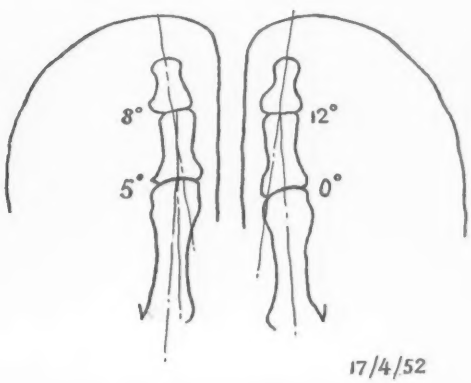

FIGURE VIIB.

After wearing "experimental" shoes for three and a half years.

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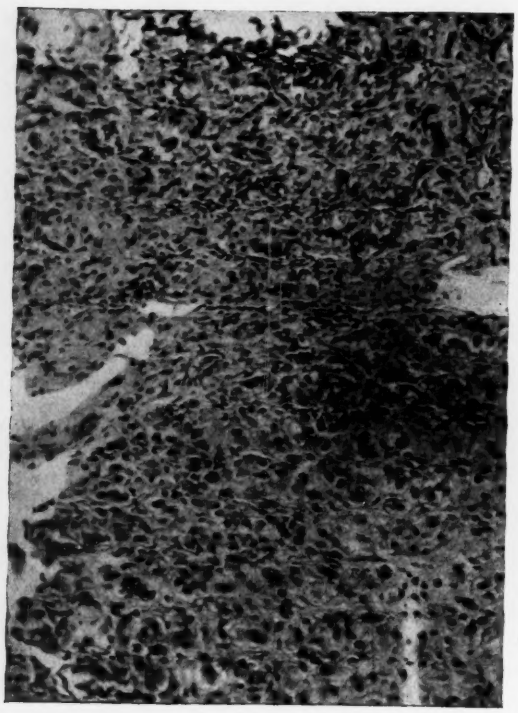

FIGURE II.

Aspiration biopsy of liver in the case of chronic brucella hepatitis reported, showing active cirrhosis with established fibrosis. There were no granulomatous lesions typical of active brucellosis in the biopsy fragment. (Hæmatoxlyin and eosin stain, × 168.)

ILLUSTRATIONS TO THE ARTICLE BY W. J. D. MIRA.

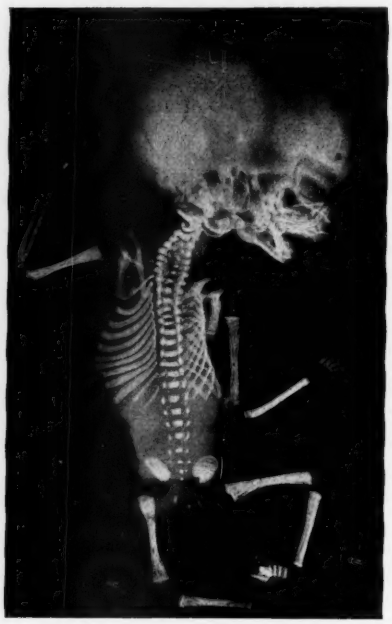

FIGURE III.

Ossification of the fœtus. Structures in the mastoid region are otic rudiments.

ILLUSTRATIONS TO THE ARTICLE BY RICHARD FLYNN AND L. D. WHEELER.

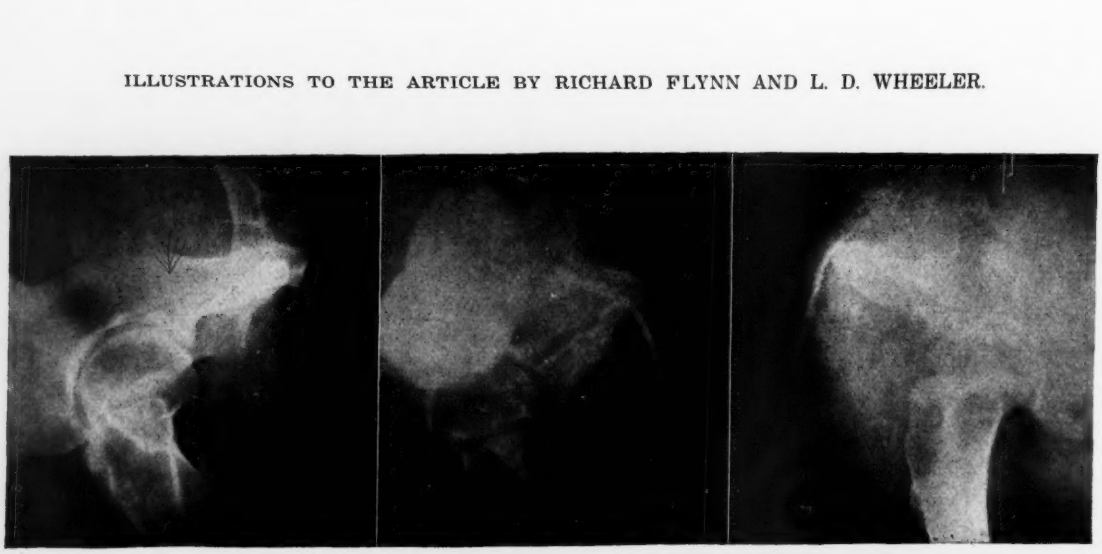

FIGURE I. FIGURE II. FIGURE III.

ventional shoes (Figure VA), then immediately in the new shoes (Figure VIA). It will be noticed that although the new shoes provide adequate space for the toes to assume their natural divergent position, they retain the convergent arrangement to a considerable extent.

By checking the external appearance of the feet against an ordinary photograph taken at the time, it was noticed that no remarkable change occurred for many months. The wearer of these shoes underwent no treatment to his feet by physiotherapy, and carried out no exercises save the spontaneous exercise that occurs in walking. It is proper to mention here that he was aged thirty-seven years, and rapid skeletal changes were therefore hardly to be expected. However, by November, 1950, some distinct change was noted, and an X-ray picture then taken showed that the toes had become less cramped. The experiment was concluded with final X-ray films taken in April, 1952 (Figure VIIA), in a pair of shoes made on the same lasts.

The most pronounced change is that seen in the position of the hallux of the right foot; its valgus tendency has been corrected and there is now a clear gap between the soft tissues of the great and second toes. The left hallux, though it had less valgus tendency originally, has not improved so much; the osteo-arthritic changes in its metacarpo-phalangeal joint already shown in the earlier X-ray films are unaltered. Spread of the smaller toes has improved only to a slight extent. Figures VB, VIB and VIIB are tracings from the foregoing X-ray films, showing the measured metatarso-phalangeal and interphalangeal valgus angles of the hallux.

Whilst the main concern of this study was with the gross objective effects obtained on the outline of the anterior part of the foot, other effects were noted, and one was unexpected.

The volunteer for this experiment had always been subject to excessive foot perspiration, and had onychocryptosis and epidermophytosis of the nail of the right hallux of several years' standing. Excessive perspiration ceased immediately in the new shoes, the epidermophytosis eventually cleared spontaneously, and the onychocryptosis, though still present to a degree, ceased to be troublesome. This unexpected benefit may be due to the fact that in these shoes the toes have more room to move, perspiration is not occluded between the toes, and maceration of the skin no longer occurs. The *hallux rigidus* on the left was a cause of pain for several years, which had become so severe on long walks that the subject was about to reconcile himself to a more sedentary existence, when he found that as soon as he started to wear the new shoes the condition became painless.

It is suggested that the medical profession might encourage the wearing of shoes of the type here described as a relief from conventional footwear, in an attempt to control the incidence of the discomfort and deformities and other conditions consequent upon the continuous wearing of badly designed footwear.

It is regretted that this work was somewhat limited in scope, and incomplete in that other possible common causes of foot distortion, such as the wearing of tight socks, were not dealt with.

Summary.

Shoes designed in accordance with the triangular plantar aspect of the natural foot were worn for a period of three and a half years.

Skeletal distortions acquired by thirty-five years' use of conventional footwear showed appreciable tendency to self-correction during this experimental period.

Painful *hallux rigidus* became painless, onychocryptosis became symptomless, and excessive foot perspiration ceased.

Acknowledgement.

The writer wishes to thank Professor H. J. Wilkinson, head of the Department of Anatomy, University of Queensland, for encouragement and help in the preparation of this paper.

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MENTAL SYMPTOMS IN POISONING WITH ATROPINE AND ITS DERIVATIVES.[1]

By I. A. LISTWAN and N. G. WHEALY,
Sydney.

ATROPINE POISONING is not infrequent, but cases in which mental symptoms dominate the picture are quite rare. It is our aim in this article to discuss the clinical syndrome with special reference to cases reported in the literature and to our own case.

Mode of Absorption.

Atropine and its derivatives may be absorbed through the gastro-intestinal tract, by subcutaneous injections, by inunction through the unbroken skin and by application to the eye in the form of eye drops.

Reviewing the recent literature with regard to mental symptoms caused by different ways of ingestion of atropine, we found the following cases.

Cases Recorded as Due to Application to the Eye.

1. Mélivier's case (1935) of a seventy-four-year-old woman showing acute confusion, excitement and hallucinations after instillation of 1% atropine eye drops.

2. Duggan's case (1937) of acute confusion after the instillation of 1% atropine eye drops.

3. Belz's case (1949) of a child intoxicated from atropine in collyrium.

4. Fleury's and Marx's case (1949) of intoxication following the use of an eye wash with atropine solution.

5. Hopkins and Robyn-Jones's case (1937) in which the symptoms appeared three days after a washout with atropine during iridectomy and were characterized by acute confusion with vivid visual hallucinations and persecutory delusions.

Cases Recorded as Due to Ingestion Through the Gastro-Intestinal Tract.

1. Hamilton and Sclare's case (1947) after the taking of 10 minims of liquid extract of belladonna equal to 1/15 grain of the alkaloid. The mental symptoms were characterized by confusion, agitation and visual hallucinations.

2. Minors's case (1948), in which five children were poisoned after eating raisin-like fruits of deadly nightshade. They became delirious, restless, grimacing and hallucinated. The symptoms appeared in four hours and lasted for two days.

3. Carter's case (1940), in which a patient swallowed five drachms of liniment corresponding to one and three-eighths grains of alkaloid. He developed mental symptoms in ten hours and recovered after spinal drainage.

4. Taylor's case (1934) of death after one drachm of liniment of belladonna had been taken by mouth by mistake.

5. Gratten's case of recovery after a wine glass of liniment had been taken.

Cases Recorded as Due to Subcutaneous Injection of Atropine.

1. Welbourn's cases (Hamilton and Sclare, 1947) after premedication with one-sixth of a grain of atropine by

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association on June 5, 1952.

error. All patients developed acute delirium with hallucinations and muscular incoordination.

2. Parfitt's 15 cases (1947) after the use of atropine in prolonged narcosis with "Dial" and paraldehyde to prevent vomiting. The dose was six minims of the liquor daily for ten days. Here the cumulative effect of prolonged small doses gave rise to the symptoms late on the sixth or eighth day. The patients showed confusion, restlessness and emotionalism and later facile cheerfulness and awkward hilarity.

In our own case the absorption occurred by application of 1% homatropine in saline to one eye on two occasions separated by four hours' interval.

Toxicology.

The usual dose of atropine is 1/150 to 1/100 of a grain when injected subcutaneously. One-thirtieth of a grain causes parasympathetic paralysis, one-twelfth of a grain causes restlessness and one-sixth of a grain mental symptoms and ataxia.

The usual dose of atropine when ingested by the gastro-intestinal tract is 10 minims of tincture corresponding to 1/300 grain of alkaloid or two to three minims of liquid extract corresponding to one-seventy-fifth of a grain of alkaloid. It is worth while to remember for practical purposes that the liquid extract is twenty times stronger than the tincture.

The usual dose of atropine when instilled into the eyes is one or two drops of a 1% solution, corresponding to 1/120 of a grain of the alkaloid. This amount usually does not cause any toxic symptoms, unless there is special susceptibility. And even so the symptoms are mostly due to parasympathetic paralysis and very rarely due to central action causing mental disturbances.

Symptomatology.

The symptomatology of atropine poisoning may be roughly divided into two groups of symptoms:

1. The peripheral symptoms due to post-ganglionic cholinergic paralysis are well known and are enumerated only in passing. They are: flushed face, dry skin, tachycardia, mydriasis with sluggish reactions, distension of bladder.

2. The mental symptoms, in which we are mostly interested, are due to central nervous system derangement: (*a*) confusional state, (*b*) vivid visual hallucinations, (*c*) restlessness, (*d*) muscular incoordination, and later (*e*) increased emotionalism.

The symptoms occur a few hours after ingestion and are transient, lasting one to three days. The patient shows a short period of retrograde amnesia and soon becomes rational and cooperative. The condition is usually not fatal and has a good prognosis. The susceptibility increases with age.

Our own case seems to be a classical one with all the enumerated symptoms present and therefore it is worthwhile to present the history in detail in order to elaborate more vividly the clinical features.

Report of a Case.

The patient was a healthy young woman with no important history of physical illness. Emotionally she could be classified as being rather on the borderline of the hypomaniacal reaction group. Her intelligence was roughly over the average and she belonged to the ideatic perception type. These characteristics of her personality together with her profession—she is an actress—could to some extent explain the picturesque syndrome she developed.

One night the patient developed suddenly an acute confusional state with the following symptoms and signs: she was confused and restless, partly undressed, getting in and out of bed and obviously having vivid visual hallucinations, shown by her persistent attempts to pick up something from the floor. She admitted later that she saw several black bugs and other little animals crawling everywhere on the floor, the wall and the curtains. She also had a marked photophobia. She was disorientated in time and place and failed to recognize members of her family. She developed an awkward hilarity, her speech was incoherent and her gait tremulous with vague gestures of physical violence.

The pulse rate was 96 per minute; respirations numbered about 30 in the minute, her face was flushed, and the pupils were dilated, the left more than the right. We could not, owing to her extreme restlessness, examine her in more detail.

In view of the sudden development and the typical syndrome of an organic state, we started to search for the drug which could cause the condition. We were told that she had developed a corneal ulcer on the left eye and seen an eye specialist in the afternoon and that he had instilled homatropine drops into the left eye at 5 p.m. and asked her to repeat it at 9 p.m. She felt dizzy when she returned home, saw one of us, was comforted, and instilled at 9 p.m. the eye drops and started to develop the symptoms two hours later.

We administered "Omnopon" 1/3 of a grain and scopolamine 1/150 of a grain and next morning she was still drowsy but completely clear and developed a retrograde amnesia. Her left pupil was still very large and the right pupil was normal. The systolic blood pressure was 135 millimetres of mercury and the diastolic pressure 70 millimetres. Her pulse rate was 84 per minute.

In the next two days she developed a peculiar emotionalism with anxiety and a tendency to crying. She settled down in about three days, but the left pupil was still dilated for about eight days. She was at this stage treated with injections of 1% pilocarpine solution.

We sent the bottle to the analyst whose report was that it contained 1% homatropine in normal saline.

We were unable to trace any idiosyncrasy in her history and the patient refused to be tested with subsequent atropine injections.

An interesting feature was that the patient's father was all his life sensitive to belladonna.

Differential Diagnosis.

The syndrome corresponds to the organic reaction type and bears most similarity to *delirium tremens*, but is of short duration.

Other possibilities are: (*a*) intoxication with "Benzedrine", (*b*) intoxication with mescaline, (*c*) intoxication with alcohol, (*d*) catatonic excitement, (*e*) maniacal phase in circular psychosis. The history, sudden onset and short duration should clarify each case. There is no specificity in the causal agent.

Treatment.

Most of the authors used "Omnopon" as a cortical depressant and also scopolamine, although scopolamine is synergistic to the atropine. However, its sedative action overweighs probably its synergistic action. Pilocarpine proved to be of no use as it antagonizes atropine only in its peripheral action. Physostigmine was used as a physiological antidote and in a few cases "Prostigmin".

Spinal drainage was applied in a few cases, stomach and rectal lavage with potassium permanganate (10 grains to a pint) and magnesium sulphate.

Summary.

The problem of mental symptoms due to atropine poisoning is discussed with special reference to the clinical syndrome.

Cases reported in the literature and a case of our own are quoted.

The atropine and its derivatives may be ingested by several routes: as an oculentum, eye drops, eye wash, subcutaneous injection, by gastro-intestinal route and through the unbroken skin.

The mental symptoms are confusional state, visual hallucinations, restlessness, and increased emotionalism. The symptoms are transient and the prognosis is good.

Finally the differential diagnosis and treatment of poisoning are mentioned.

Acknowledgement.

We wish to express our thanks to Miss J. Ashby, librarian of Sydney Hospital, for her assistance in collecting scattered references on this not very clear-cut subject.

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RELATIONSHIP BETWEEN HUMAN INFECTION WITH BRUCELLOSIS AND A HEAVILY INFECTED MILK SUPPLY.

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THERE have been a number of reports of human infection in selected populations exposed to particular risks of infection with *Brucella abortus bovis.* Thus infection in dairy workers (Cruickshank and Barbour, 1931), cattle attendants (Thomsen, 1931), veterinary surgeons (Wilson, 1932; Thomsen, 1931) and abattoir workers (Dible and Pownall, 1932) has been described; but so far as can be ascertained, little is known about the rate of infection in a normal population known to be consuming, and to have consumed for some time, infected milk.

In the course of a wider survey of the incidence of *Brucella abortus* infection in milk, a small area was encountered where all the liquid milk supplies came from one retailer. This retailer obtained his milk from four separate farms. Each of these was examined for the incidence of brucella infection in the cattle and milk.

The cattle were examined by testing blood samples for brucella agglutinins and the milk by the "ring" and "whey" tests. In addition milk samples from all cattle giving positive results to blood tests were further tested by guinea-pig inoculation.

Of the four herds supplying milk to this retailer, three were found to be clear of infection and one to be heavily infected. In this last herd of 25 cows, 16 milk samples gave positive results to the "ring" test and 14 to the "whey" test. Brucella agglutinins were present in the blood of 19 cows, while the organism was recovered from the milk of six. All six strains gave the typical cultural reactions of *Brucella abortus bovis.*

After the list of customers provided by the retailer had been consulted, it was found that milk from each herd was supplied to separate sets of consumers. It was therefore decided to visit a representative sample of consumers containing both those receiving milk from the infected herd and those receiving milk from "clean" herds. The former group was visited first, and in view of the small number of positive reactions no visits were made to those not receiving infected milk.

The medical practitioner, Dr. A. D. Reid, gave permission to approach his patients and take blood samples from those willing to cooperate. This was done and 45 samples were collected. In only two instances did the person approached refuse. Of these 45 people, 18 always drank unboiled milk, while two always boiled it before drinking it. The remaining 25 usually boiled their milk during the hot weather, but not during the cooler months.

The serum from each person was examined by the following four tests: (*a*) direct agglutination, (*b*) a modified Coombs test, (*c*) a "blocking" test, (*d*) guinea-pig inoculation.

Direct Agglutination.

For the direct agglutination test, serial doubling dilutions of serum ranging from one in 10 to one in 640 were used.

Modified Coombs Test.

The modified Coombs test, first described by Coombs (1945), is an antiglobulin test for the detection of weak or incomplete antibodies. The method used was as follows. Tubes showing no agglutination in the direct agglutination test were centrifuged and the deposits washed three times, approximately 0·8 millilitre of saline being used each time. The washed deposit was then emulsified in 0·5 millilitre of saline, and one drop of rabbit anti-human globulin serum (Proom, 1943) was added. The tubes were then incubated overnight at 37° C., and the results were read the next morning as for a direct agglutination test.

"Blocking" Test.

The "blocking" test was carried out as follows. One drop of a one in 40 dilution of a specimen of serum with a known brucella agglutinin titre of one in 1280 was added to tubes showing no agglutination in the direct agglutination test. They were then incubated overnight at 37° C. This quantity of serum always produced agglutination in the saline control of the direct test; but if the unknown serum contained "blocking" antibodies, this addition of "positive" serum failed to cause agglutination. If agglutination occurred, then "blocking" was not taking place.

Guinea-Pig Inoculation.

Guinea-pig inoculation was carried out as follows. After removal of the serum, the blood clots were ground up with silica and saline in sterile mortars and two millilitres of each suspension were injected subcutaneously into guinea-pigs. The guinea-pigs were killed and examined for brucella infection five to six weeks after inoculation.

Results.

A total of 45 specimens of serum were examined, of which only one gave a positive response to the direct agglutination test, and four gave a positive result to the modified Coombs test; no evidence of the presence of blocking antibodies was obtained. All tests were controlled by a serum procured from Melbourne. This serum failed to react to the direct agglutination test, but gave a positive result with the modified Coombs test and showed the presence of blocking antibodies. Cultures and blood agglutination tests from all the guinea-pigs gave negative results.

The only serum giving a positive result to the direct agglutination test when the titre was one in 80 was taken from a young married woman who had a baby seven or eight weeks old. She had consumed a large quantity of raw milk during pregnancy and had not felt well enough to leave hospital until three weeks after the birth of the baby. With this serum a fourfold increase in titre (one in 320) was obtained with the modified Coombs test.

Discussion.

It is well known that in general the number of clinical cases of brucellosis due to *Brucella abortus bovis* in man

is small compared with the wide distribution of contagious abortion in cattle. Most of the known cases are in groups exposed to infection through abraded skin, and the results reported above confirm the suggestion, based on the epidemiology of the disease, that man is relatively resistant to infection by ingestion. At the most we were able to find only one doubtful (undiagnosed) clinical case and three doubtful cases of subclinical infection in a series of 45 people, all but two of whom were exposed to repeated infection by drinking unboiled milk containing *Brucella abortus* of normal virulence to cattle.

This work therefore supports the view that *Brucella abortus* infection in man does not depend mainly on the consumption of infected milk, but rather on other methods of infection or on secondary factors at present unknown.

Acknowledgements.

Special thanks are due to Dr. J. A. R. Miles and Sister E. I. Uren for their assistance in collecting blood samples, and also to Dr. M. M. Wilson, of the University of Melbourne, for supplying the control serum.

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CORONARY ARTERY DISEASE IN WESTERN AUSTRALIA, 1937 TO 1950.

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A STATISTICAL analysis of coronary artery disease, as it is recorded in the Statistical Register of Western Australia from 1937 to 1950, has been made in an attempt to ascertain the true increase in this disease.

Morris (1951), investigating 6000 necropsy reports from London Hospital between 1907 and 1949, draws the following conclusions: (i) The increase in coronary heart disease over that period has been sevenfold. (ii) As a common disease it is new and not newly recognized. (iii) Coronary atheroma has decreased during that period, and major periods of decrease were connected with the two World Wars.

The apparent contradiction between (i) and (iii) may largely be explained by the fact that atheroma was "defined mainly by report of calcified arteries". Atheroma cannot be measured by calcification.

Buck *et alii* (1950), in describing atheroma in cerebral arteries, make the following statement: "The concentration of calcium does not increase with the degree of atherosclerosis. In other words, cerebral arteries do not usually calcify."

Cleland (1949), in a paper based on 6000 tabulated post-mortem examinations at the Royal Adelaide Hospital between 1920 and 1946, together with 600 autopsies at mental hospitals and 250 autopsies performed for the coroners during the war years, concludes, among other things, that infarction of heart muscle from coronary disease has increased from 11 per 1000 in the period 1920-1925 to 45 per 1000 in the period 1943-1946, that the increase is greater than can be explained by aging of the population, and that it cannot be explained by better post-mortem diagnosis.

Ryle and Russell (1949) in a clinical and epidemiological study of coronary disease based on a statistical analysis of material to be found in the reports of the Registrar-General (United Kingdom) for a twenty-five year period and of cases encountered by Russell in consulting practice over a similar period, make the following statements: (i) The mortality amongst males and females aged thirty-five years and upwards in 1945 was 15 times greater than in 1921. (ii) There was evidence that in the age period forty to fifty-five years, the male death rate was increasing more rapidly than the female death rate. (iii) The increase in the coronary disease mortality in the post-war period is unlikely to be wholly due to transference of myocardial deaths.

TABLE I.

Mortality Rate per 1000, Males and Females, 1938 to 1950.

Year.	Coronary Artery Disease and *Angina Pectoris.*		Aortic Aneurysm and Pulmonary Embolism.	
	Male.	Female.	Male.	Female.
1938	0·61	0·26	0·05	0·03
1939	0·67	0·38	0·08	0·03
1940	0·76	0·32	0·09	0·04
1941	0·84	0·28	0·08	0·05
1942	0·88	0·29	0·05	0·03
1943	0·86	0·44	0·05	0·03
1944	0·90	0·45	0·06	0·03
1945	0·87	0·43	0·07	0·04
1946	1·05	0·46	0·11	0·06
1947	1·18	0·53	0·07	0·06
1948	1·24	0·50	0·07	0·09
1949	1·34	0·58		
1950	1·47	0·67		

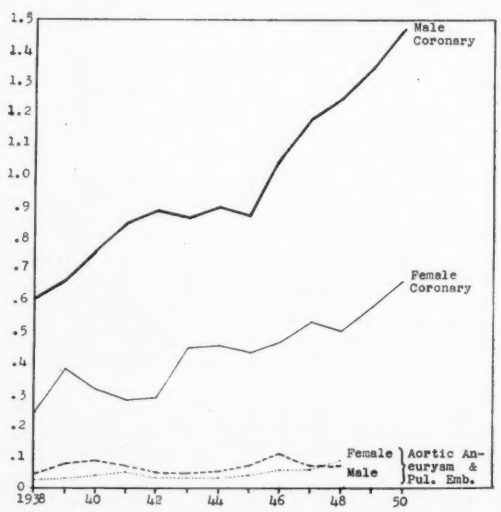

FIGURE I.

Crude mortality rates, male and female: coronary artery disease, aortic aneurysm and pulmonary embolism.

The increase from 1938 to 1950 in coronary artery disease in Western Australia is indicated in the increase in the mortality rate for that disease in Table I and Figure I (male increase 141%, female increase 120%).

The main purpose of the present investigation is to ascertain how much of this increase is real and not due to

TABLE II.

Age Distribution of Deaths from Coronary Artery Disease Compared with Deaths from All Causes in Various Age Groups.

Age Group (Years).	1937–1939.			1947–1949.		
	1 Coronary Deaths.	2 Deaths from All Causes.	3 Percentage 1 of 2.	4 Coronary Deaths.	5 Deaths from All Causes.	6 Percentage 4 of 5.
Males:						
20 to 29	1 (0·2%)	365	0·28	0 (0%)	240	0
30 to 39	8 (1·8%)	434	1·84	8 (0·8%)	256	3·12
40 to 49	27 (6%)	543	4·97	76 (7·4%)	585	13·0
50 to 59	79 (18%)	1000	7·9	203 (20%)	1061	19·13
60 to 69	175 (40%)	1722	10·16	322 (31·6%)	1727	18·64
70 and over	150 (34%)	2658	5·64	409 (40·2%)	3653	11·2
Total	440 (100%)	6722	6·54	1018 (100%)	7522	13·53
Females:						
20 to 29	1 (0·5%)	215	0·47	1 (0·3%)	121	0·83
30 to 39	4 (2%)	275	1·46	0 (0%)	205	0
40 to 49	18 (9·4%)	364	4·94	18 (5·5%)	385	4·67
50 to 59	27 (14·1%)	686	3·94	41 (12·5%)	562	7·3
60 to 69	65 (34%)	959	6·78	122 (37·3%)	998	12·22
70 and over	76 (40%)	1694	4·49	145 (44·4%)	2766	5·24
Total	191 (100%)	4193	4·55	327 (100%)	5037	6·49

the influence of an aging population or to a change in fashion of diagnosis.

Table II compares the two three-year periods, 1937 to 1939 and 1947 to 1949, to show the increase in the various age groups of death from coronary artery disease and death from all causes.

The coronary deaths in each group are shown as a percentage of total coronary deaths and as a percentage of deaths from all causes in the group.

Figure II compares the latter for males and females in the two periods reviewed.

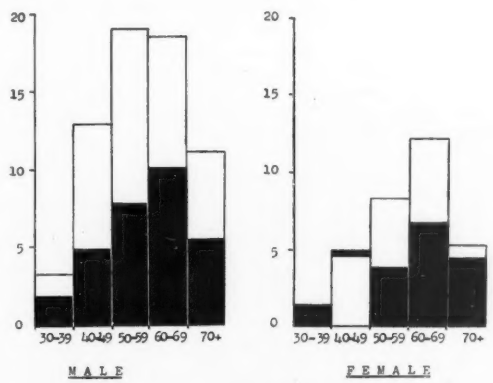

FIGURE II.

Percentage of coronary deaths to deaths from all causes in age groups; solid columns, 1937 to 1939; open columns, 1947 to 1949.

Table III, Part I, compares the coronary deaths in age groups for consecutive three-year periods by giving the mortality rate in the group and the amount of increase of this rate from the first three-year period—that is, the comparative mortality index.

Table III, Part II, gives similar details for myocarditis and other diseases of the heart.

The mortality rate and the comparative mortality index for age groups are illustrated by plotting the mortality rates on semilogarithmic grids (Figure III).

In Table IV, standardized death rates are given from 1937 to 1949 for coronary disease and myocarditis and other heart diseases as far as they concern the population between the ages of twenty and sixty-nine years. The means and regression coefficients are also shown, and by their use the standardized death rates are shown graphically as lines to their regression equations on Figure IV. The total standardized death rate for coronary disease *plus* myocarditis *et cetera* is also shown in the same manner on the graph.

TABLE III.

Mortality Rate per 1000 (M.R.) and Comparative Mortality Index (C.M.I.).

Age Group. (Years.)		1937 to 1939.	1940 to 1942.	1943 to 1945.	1946 to 1948.
Part I: Coronary Artery Disease.					
Males:					
40 to 49.	M.R. ..	0·31	0·45	0·45	0·61
	C.M.I. ..	1·0	1·45	1·45	1·97
50 to 59.	M.R. ..	1·13	1·89	1·66	2·51
	C.M.I. ..	1·0	1·67	1·47	2·22
60 to 69.	M.R. ..	3·42	4·06	4·19	5·31
	C.M.I. ..	1·0	1·19	1·23	1·55
70 and over.	M.R. ..	5·12	6·72	7·49	9·54
	C.M.I. ..	1·0	1·31	1·46	1·86
Females:					
40 to 49.	M.R. ..	0·23	0·06	0·13	0·15
	C.M.I. ..	1·0	0·26	0·57	0·65
50 to 59.	M.R. ..	0·44	0·41	0·56	0·62
	C.M.I. ..	1·0	0·92	1·27	1·41
60 to 69.	M.R. ..	1·49	1·48	1·68	2·2
	C.M.I. ..	1·0	0·93	1·13	1·48
70 and over.	M.R. ..	3·05	3·96	5·28	5·23
	C.M.I. ..	1·0	1·3	1·73	1·72
Part II: Myocarditis and Other Diseases of the Heart.					
Males:					
40 to 49.	M.R. ..	0·56	0·35	0·39	0·31
	C.M.I. ..	1·0	0·63	0·7	0·55
50 to 59.	M.R. ..	1·85	1·43	1·95	1·92
	C.M.I. ..	1·0	0·77	1·05	1·04
60 to 69.	M.R. ..	5·42	6·59	6·57	6·25
	C.M.I. ..	1·0	1·22	1·21	1·15
70 and over.	M.R. ..	18·35	19·51	22·95	26·24
	C.M.I. ..	1·0	1·06	1·25	1·43
Females:					
40 to 49.	M.R. ..	0·4	0·47	0·31	0·26
	C.M.I. ..	1·0	1·17	0·77	0·65
50 to 59.	M.R. ..	0·99	0·96	1·09	1·07
	C.M.I. ..	1·0	0·97	1·1	1·08
60 to 69.	M.R. ..	3·49	4·23	4·09	3·34
	C.M.I. ..	1·0	1·21	1·17	0·97
70 and over.	M.R. ..	16·53	17·19	18·77	19·55
	C.M.I. ..	1·0	1·04	1·14	1·18

In Table I and Figure I deaths from aortic aneurysm and pulmonary embolism have also been shown.

Discussion.

There is no evidence in Table I and Figure I of transference of diagnosis from aortic aneurysm and pulmonary embolism to coronary artery disease. There is no fall in the latter to offset the increase of the former. Indeed, cases of aortic aneurysm and pulmonary embolism have from the one disease for a rise in the other. This is most pronounced in the males.

The increase in coronary disease in the seventy and over age groups shown in Tables II and III is very suspect.

Since the 1938 revision of the Statistical Register, the Registrar-General has increased the emphasis on the unsuitability of such undesirable terms on a death certificate as senility, heart failure, myocardial failure *et cetera*, and insists on a diagnosis of some specific disease or heart

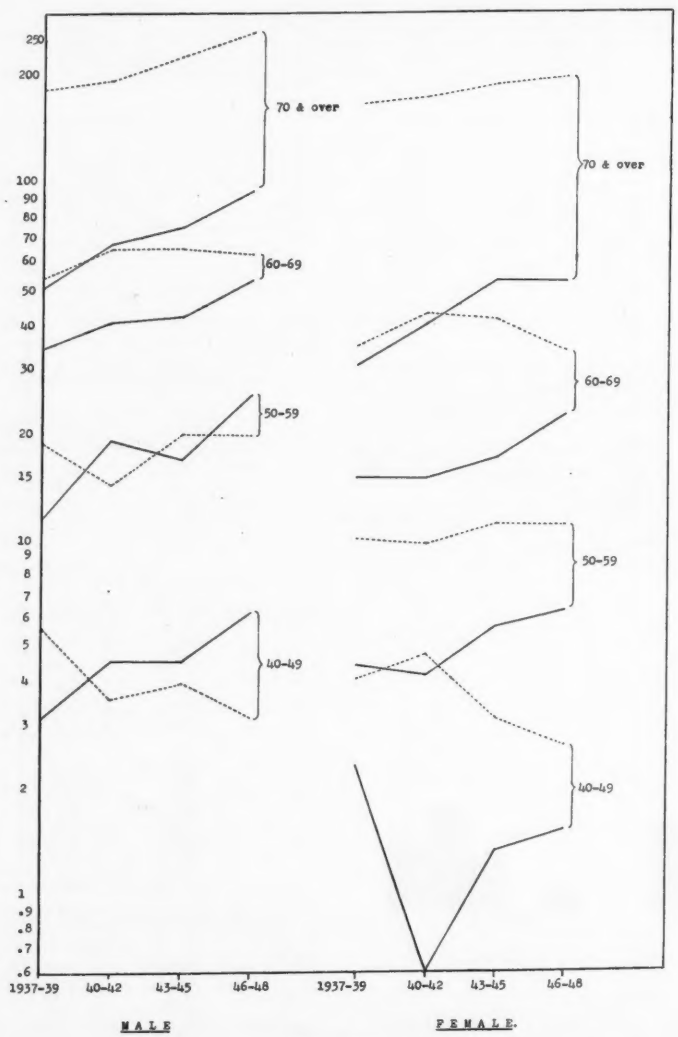

FIGURE III.

Mortality rate in age groups for coronary artery disease and combined myocarditis and other diseases of the heart in triennia from 1937 to 1948, shown on semilogarithmic grid to display rates of change. Continuous line, coronary artery disease; interrupted line, myocarditis *et cetera*.

beginning of the period reviewed been so few that they can have little influence on coronary artery disease rates, transference or no transference.

Figure III, however, gives somewhat dramatic evidence of the possibility of such transference between myocarditis and other diseases of the heart, and coronary artery disease. If the seventy and over age groups are disregarded, the majority of the groups show a compensatory fall in disease. The result is that the practitioner faced with the certification of a person in extreme old age who has died from the normal physiological coronary circulatory insufficiency of the aged, is in some quandary regarding the "disease" causing death and very naturally puts it in the nearest category, that of coronary occlusion. This increasing tendency obscures the whole picture if the aged are included in our total figures, as no differentiation is made

TABLE IV.

Standardized Death Rates in the Population Twenty to Sixty-Nine Years of Age.

Year.	Population 20 to 69 Years.		Coronary Artery Disease.				Myocarditis and Other Diseases of the Heart.			
			Number of Deaths.		Standardized Death Rate per 1000.		Number of Deaths.		Standardized Death Rate per 1000.	
	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.
1937	151,731	130,509	78	33	0·53	0·26	141	81	0·95	0·64
1938	152,572	132,048	95	36	0·64	0·28	177	86	1·19	0·67
1939	153,513	133,733	117	46	0·78	0·35	172	91	1·14	0·69
1940	154,656	135,551	124	38	0·82	0·28	132	81	0·87	0·61
1941	155,907	137,439	127	32	0·83	0·23	173	84	1·13	0·69
1942	151,310	137,512	145	36	0·96	0·26	188	128	1·24	0·92
1943	152,666	139,197	127	45	0·83	0·32	177	98	1·16	0·71
1944	153,822	140,905	128	48	0·83	0·33	161	84	1·04	0·59
1945	155,056	142,647	138	40	0·88	0·28	201	124	1·29	0·86
1946	156,235	144,356	178	56	1·13	0·38	193	92	1·23	0·63
1947	157,168	146,108	174	56	1·08	0·37	150	101	0·94	0·67
1948	160,322	149,597	201	61	1·23	0·4	186	85	1·14	0·56
1949	169,552	155,886	234	65	1·37	0·41	170	97	1·0	0·62
	Mean				0·92	0·32	—	—	1·1	0·68
	Regression coefficient				0·055	0·011	—	—	0·0025	0·0037

between the pathological atheromatous occlusion of the younger groups and the physiological sclerosis of the aged.

For this reason, the seventies and over were left out of Table IV. The under-twenties were also left out, as they are little influenced by the disease.

It will be seen in Table IV and Figure IV that the total figures give less evidence of transference between the diseases, particularly in the males, among whom there has been a slight increase in myocarditis *et cetera* in addition to the large increase in coronary disease.

The 1943 population was used as a standard. The standardized death rate indicates the death rate that would have been recorded in the year in question if it had had a population identical with that of 1943. By this means the influence of an aging population is removed.

The true increase in coronary disease during the thirteen years reviewed is, therefore, somewhere between the coronary regression line and the combined coronary and myocarditis line—that is, in males less than 112% and more than 41% and in females less than 52% and more than 9%.

TABLE V.

Annual Consumption in Pounds of Butter and Tobacco per Head of Population in Three-Year Periods.

July to June.	Butter.	Tobacco.
1936 to 1939 ..	31·18	3·93
1942 to 1945 ..	28·8	3·8
1946 to 1949 ..	23·49	4·57

Table III indicates that the increase has been most rapid in the male age groups forty to fifty-nine years, but that the indications for transference are also greatest in these groups. The fifty to fifty-nine age group of males, however, in spite of the transference, shows an increase in myocarditis *et cetera* in addition to the increase in coronary disease.

Table II shows how the increase in coronary disease may appear exaggerated. There has been a very slight increase in the proportion of coronary deaths in the forty to fifty-nine years groups; but, owing to the greatly decreased total death rate in these groups, they now form a very substantial part of the deaths in these groups. That is, the practitioner meets them relatively more frequently, not only because of the small proportionate increase in the groups, but because of the many fewer deaths from other causes occurring in these groups.

The transference, then, has merely slowed down the rate of increase of myocarditis *et cetera* and accelerated the increase in coronary disease. We cannot obtain much reassurance from this because of the over-all increase in the two groups, however the diagnosis is made. This increase is shown in the top line of the male and female graphs of the regression lines.

War Period.

Closs and Dedichen (1949) have mentioned the recession in the coronary mortality rate in other countries during the war years. It has been suggested that the restricted diet of that period may have been an influence, particularly the restriction of fats.

In Western Australia the rationing of butter to six ounces per week and the absence of rationing of other fats could hardly be termed a restriction in a physiological sense.

On the other hand, tobacco was scarce and difficult to come by in quantities sufficient to satisfy a heavy smoker.

Table V and Figure V show the annual consumption of butter and tobacco per head of population in three-year periods.

It will be seen that the graph of butter consumption bears no relation to that of coronary artery disease, but the tobacco consumption follows that disease very closely. Whether this is coincidence or not is a matter for further investigation.

The method of recording the statistics may, however, be a factor in inducing a recession of the coronary curve for the war years.

The Registrar-General has not adjusted his population figures for movement of troops and the deaths do not include those of service personnel. There is a possibility, therefore, that our recorded population is a little too high and that the deaths are too low for the war years, which would account for the recession.

It is, however, rather unexpected that the recession is most pronounced in males aged fifty to fifty-nine years, a group that would not be greatly affected by war service. It seems likely that the well recognized psychological factor of mental stress and strain is an influence; but our graph would indicate that the danger period is not during the period of such strain, but in the period of relaxation that follows. In other words, "doctor's disease" is not the disease of the doctor struggling to establish himself in practice, but of the doctor who has so struggled and has just reached the stage of professional security and success.

Statistical Data.

Aortic aneurysm and pulmonary embolism do not appear in the Statistical Register, for the years under review, as separate diseases. The figures were therefore acquired by the simple but laborious process of examining some 50,000 death certificates in the Government Statistician's files.

Coronary artery disease is recorded as for that disease and *angina pectoris* under rubric 94 of the register. Myocarditis and other diseases of the heart are the deaths given in rubrics 93, 90 and 95 of the register.

No adjustment has been made for the 1938 revision of classifications, as little difference to the figures arose thereby. The 1950 figures are those obtained under the 1938 revision. They were not available in separate age groups and so have been omitted from age group tables. The 1948 classification adopted for the 1950 register materially affects the transference from myocarditis to coronary disease, and in future considerable adjustment will be required to compare mortality rates before and after 1950. By the 1938 classification coronary deaths for 1950 are 394 among males and 190 among females; the 1948 revision makes these figures 470 and 237 respectively.

The figures given for the wartime consumption of butter and tobacco cannot be considered as very accurate, as no account is taken of troop movements and import and export of military stores. However, they have both been computed by the same method.

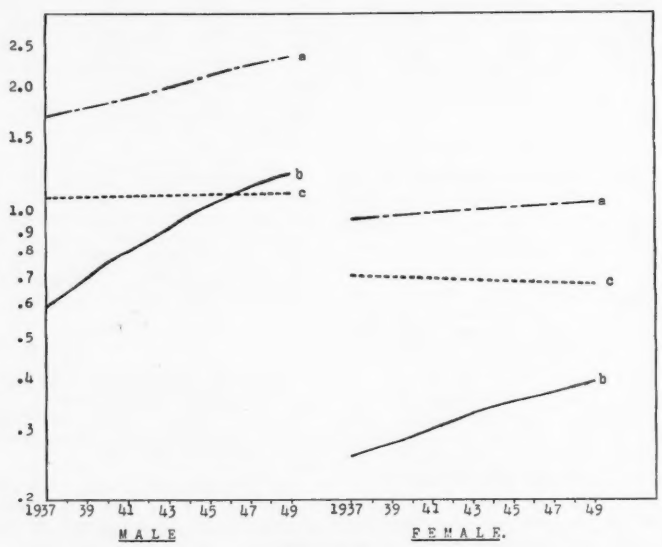

FIGURE IV.

(*a*) Combined coronary disease, myocarditis and other diseases of the heart. (*b*) Coronary artery disease. (*c*) Myocarditis and other diseases of the heart. Progression of standardized death rates from 1937 to 1949 shown on semilogarithmic grid to display comparative degree of change.

Summary and Conclusion.

A statistical analysis of deaths from coronary artery disease, myocarditis and other diseases of the heart, as given in the Statistical Register, and of aortic aneurysm and pulmonary embolism from scrutiny of death certificates between the years 1937 to 1950, gives rise to the following deductions.

1. The true increase in coronary artery disease is not so great as indicated in crude mortality rate tables, nor so great as that suggested by some other writers for other States or countries.

2. A change of fashion in diagnosis and an aging population make the increase appear greater than it really is.

3. The true increase for males from 1937 to 1949 is somewhere between 41% and 112% and for females between 9% and 52%.

4. The true increase is largest in the fifty to fifty-nine years age group in males.

5. There is need for differentiation on death certificates between coronary artery occlusion associated with pathological atheroma and coronary circulation insufficiency from generalized arteriosclerosis of old age.

6. There is no indication of a lower fat content in wartime diet in association with the recession in coronary disease increase during that period. The consumption of tobacco, however, followed the curve of coronary disease.

7. The suggestion is made that the danger period is not during the time of mental strain, but in the period of relaxation immediately following it.

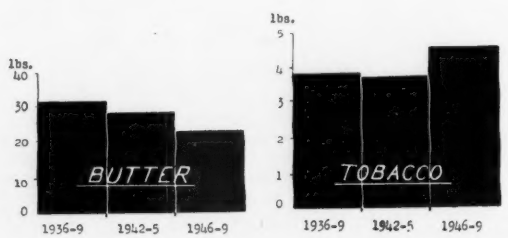

FIGURE V.

Annual consumption per head of population of butter and tobacco.

Acknowledgements.

This investigation was undertaken at the request of the State Health Council of Western Australia, and is published with permission of the Commissioner of Public Health. Acknowledgements are due to the Registrar-General and Government Statistician and his staff for assistance in

obtaining statistical data, and to Mr. A. C. Waldon, of the Department of Public Health, for his preparation of the graphs.

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Reports of Cases.

CHRONIC BRUCELLA HEPATITIS WITH SUPPURATIVE BRUCELLA PAROTITIS OCCURRING AFTER ANTIBIOTIC THERAPY.

By R. A. JOSKE[1] AND E. L. FRENCH.[2]

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DESPITE the advent of antibiotics and chemotherapy in the treatment of chronic brucellosis, the results of treatment in this disease are not yet good, and recurrences are only too common. The following case is reported primarily because it sheds some light on the pathological mechanism of such relapses. It is also of interest for other reasons —it illustrates the use of antibiotic therapy in chronic non-suppurative hepatitis, and the difficulties of bacteriological and serological diagnosis of brucellosis. Lastly, the occurrence of suppurative brucella parotitis does not seem to have been previously reported in the literature.

Clinical Record.

B.B., a male patient, aged fifty-eight years, was first admitted to the Clinical Research Unit of the Royal Melbourne Hospital on February 21, 1950. In 1916 he had an attack of fever in Malta, which resolved uneventfully. Seven years before his admission to the Clinic he had two bouts of pain suggestive of biliary colic. Nine months before his admission he began to suffer from tiredness and lassitude, and seven months later from cramping pains in his limbs, anorexia, fever and night sweats. He had lost five pounds in weight, but his diet was adequate.

On examination, the patient was florid and slightly icteric, with spider nævi and scattered petechiæ on his skin. His temperature was 98·4° F. and his pulse rate 92 per minute. His liver edge was palpable two centimetres below the right costal margin, and his spleen one centimetre below the left costal margin. A moderate degree of ascites was present.

Liver function tests confirmed the presence of chronic non-suppurative hepatitis (Figure I). Blood examination gave the following results. The hæmoglobin value was 14·6 grammes per 100 millilitres and the leucocytes numbered 3900 per cubic millimetre. Agglutination tests against *Brucella abortus* gave positive results to a dilution of one in 1280. Blood culture produced no growth of microorganisms. Liver biopsy (King and Perry, 1948) revealed active cirrhosis, but no granulomatous lesions suggestive of brucellosis were observed (Figure II).

His condition rapidly improved with rest and dietetic therapy, the ascites resolving and his temperature falling to normal. He was discharged from hospital on March 14.

He was readmitted to the clinic on June 26, 1950, complaining of lassitude, tiredness, sleeplessness and evening fever and sweats. The clinical findings were little changed, although no ascites was detectable on this and subsequent occasions.

He received an oral course of aureomycin, 750 milligrammes every eight hours for fourteen days. This produced a good clinical and subjective response, although there was little change in the biochemical findings (Figure I). He was discharged from hospital on July 14.

During the next months his progress was observed periodically in the out-patient department. His tiredness and evening fever persisted, his appetite was poor, and he was mentally depressed. His serum still agglutinated *Br. abortus* to a titre of one in 320 to one in 640.

He was admitted to hospital for the third time on January 22, 1951. The clinical and biochemical findings were little changed. An intensive course of chemotherapy was given. This consisted of six grammes of "Sulphatriad", and 0·5 gramme of streptomycin daily for twenty-one days, and 2·0 grammes of aureomycin given orally for fourteen days. There was again clinical and subjective improvement, and he was discharged from hospital on February 13. Two days later a painless lump developed in his right parotid area. This did not alter in size in relation to meals, and an X-ray examination revealed no calculi. The mass was aspirated twice in the course of four months and pus was obtained from which *Br. abortus* was isolated in pure culture on each occasion. However, after each aspiration it again slowly increased in size to a maximum of four centimetres in diameter. The abscess was aspirated a third time on July 12, 1951, and a solution containing aureomycin (250 milligrammes), streptomycin (250 milligrammes) and terramycin (250 milligrammes) was instilled. This resulted in an acute inflammatory change, necessitating a further admission to hospital on July 17.

On examination of the patient at this time, there was a fixed, tender, fluctuant mass, measuring four centimetres by three centimetres by three centimetres, lying anterior to his right ear. The overlying skin was stretched but not reddened. There was no regional lymphadenitis. His temperature was normal. Brucella agglutination tests produced negative results, although the antiglobulin test (Wilson and Merrifield, 1951) produced a positive result to a dilution of one in 5280. There was a slight improvement in the results of his liver function tests. The white cells numbered 5350 per cubic millimetre.

An oral course of terramycin, 2·0 grammes daily, was commenced, and operative drainage of the abscess was performed by Dr. J. O. Smith on July 19. The pus obtained at operation was sterile. The patient's post-operative course was uneventful, and terramycin therapy was discontinued on August 2. His subsequent condition has remained good, there being no further fever or sweats, and the results of his liver function tests continue to improve.

Isolation and Identification of the Organism from the Pus.

The first specimen of pus aspirated from the swelling was plated on nutrient 5% horse blood agar and incubated in a Mackintosh and Fildes jar, in which 20% of the air had been replaced by equal parts of oxygen and carbon dioxide. The jar was transferred to the 37° C. incubator, and after three days' incubation a heavy growth of greyish, smooth colonies up to one millimetre in diameter was obtained.

In stained preparations this organism was found to be a small Gram-negative cocco-bacillus. It was non-motile when grown in tryptone broth ("Difco") and required an increased carbon dioxide tension for growth; repeated attempts at simple aerobic subcultivation failed.

A saline suspension was readily agglutinated in a slide agglutination test with *Br. abortus* and *Br. melitensis* antisera, but *Salmonella typhi* and *S. paratyphi B* (Commonwealth Serum Laboratories) failed to agglutinate

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the organisms. The slide test result was confirmed when it was found that a saline suspension of the isolated organism was agglutinated to full titre by the *Br. abortus* antiserum.

The complete dependence on an increased carbon-dioxide tension for growth, the agglutination with brucella antiserum and the morphology and staining properties of the isolated organism seemed to indicate that it was almost certainly a strain of *Br. abortus*. However, it is more common for *Br. melitensis* to give rise to subcutaneous abscesses, *Br. abortus* rarely being found in this role. *Br. melitensis* probably does not occur in Australia; the only subjects of infection with this organism seen in Australia have been exposed to infection in the Mediterranean area. *Br. abortus* on the other hand is widespread in the dairy herds of Australia, and brucellosis always has to be considered in the differential diagnosis of any pyrexia in a patient. *Br. suis* has been isolated on occasions from diseased pigs in Queensland and New South Wales (Sutherland, 1950). Up to the present no isolation of this species has been recorded from a human case in Australia, but Derrick and Brown (1950), on epidemiological grounds, considered that three of their cases were due to *Br. suis*.

These epidemiological considerations made the identification of the organism isolated from this case of some considerable importance. It was therefore decided to send the organism to Dr. K. F. Meyer, of the Hooper Foundation in San Francisco. Dr. Meyer kindly examined the organism and reported as follows:

The culture gave the following reactions:

Requires CO_2 for growth.

Gram-negative, non-motile minute bacillus.

Produces H_2S ++++.

Growth inhibited in dilutions of 1:50,000 through 1:100,000 of thionin.

Grows in the presence of fuchsin in dilutions 1:50,000 +++, 1:75,000 ++++, and 1:100,000 ++++.

Production of ammonia from urea in one hour.

Falls into the bovis-porcine group by the adsorption test.

The culture is therefore a strain of *Br. abortus*.

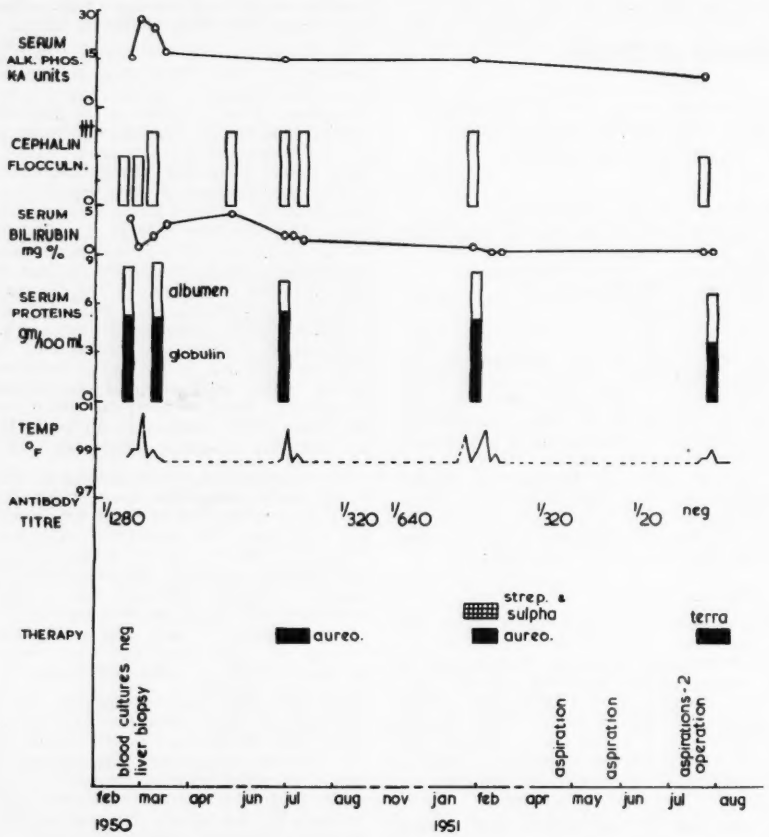

FIGURE I.

Biochemical and serological findings in the case of chronic brucella hepatitis reported. The biochemical findings are characteristic of chronic hepatitis.

Discussion.

Several aspects of this case merit further discussion.

1. The source of infection, which is unknown. Because of the patient's past history of a febrile illness while in Malta, it was thought possible that the infection might have been due to *Br. melitensis*. However, the organism recovered was shown to be a strain of *Br. abortus*.

2. The serological findings. When the patient was first examined, brucella antibody was present at a titre of one in 1280. The titre slowly fell over the succeeding months, and no antibody was present on two occasions eighteen months later at the time when isolation of *Br. abortus* was possible. However, at this time the anti-globulin test (Wilson and Merrifield, 1951) demonstrated antibody at a titre of one in 5280, although it could not be found by ordinary techniques.

3. The coexistence of chronic non-suppurative hepatitis. The ætiology of this hepatitis is of interest. The patient had had an adequate diet and there was no history of previous blood or serum transfusions, or of acute infectious hepatitis. Liver biopsy revealed no granulomata suggestive of brucellosis. Nevertheless, McCullough and Eisele (1951) give details of a case in which it was possible to show histologically the development of a nodular regenerative cirrhosis from acute granulomatous brucella hepatitis. These authors also briefly review the literature of chronic hepatitis due to brucellosis. Their paper leaves little doubt that chronic non-suppurative hepatitis may occasionally be due to brucellosis. It appears probable that the brucellosis is ætiologically related to the chronic hepatitis in the present case. A search for evidence of brucellosis in cases of chronic non-suppurative hepatitis with hyperglobulinæmia, by means of the technique of Wilson and Merrifield (1951), would be of interest. Shaffer *et alii* (1950) first reported benefit from aureomycin therapy in chronic non-suppurative hepatitis. In the case here reported two courses of aureomycin were administered without significant change in liver function tests. Some improvement was observed during the period of observation, but this did not occur concurrently with aureomycin therapy, and is considered to be due rather to the alleviation of the *Br. abortus* infection.

4. The persistence of brucella infection after antibiotic therapy. With the advent of antibiotic therapy, the results of treatment of chronic brucellosis have been vastly improved, although relapses still occur. In the present case relapse occurred even after full courses of aureomycin, streptomycin and "Sulphatriad". The later history of this case suggests that such relapses are due to localization of infection at sites not permeated by antibodies or by antibiotics administered orally or parenterally. The persistence of a high brucella antibody titre in the serum makes reinfection seem a less likely explanation of relapse. In such cases with localized lesions, it has been recommended (Herrell, 1949) that three grammes of aureomycin and one gramme of streptomycin be administered daily for twenty-eight days. The site of localization in the parotid gland, resulting in suppurative parotitis, is also of interest. Suppurative brucella parotitis does not appear to have been previously reported in the literature.

Summary.

A case of chronic hepatitis probably due to *Br. abortus* infection is reported. Suppurative parotitis, from which *Br. abortus* was isolated in pure culture, occurred after two courses of aureomycin, and despite a high titre of antibody against *Br. abortus*.

Acknowledgements.

Our sincere thanks are due to Dr. K. F. Meyer, of San Francisco, for permission to quote his bacteriological findings, and to the staff of the Royal Melbourne Hospital who have so generously helped us in this study.

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FŒTUS PAPYRACEUS: AN UNUSUAL CASE.

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FŒTUS PAPYRACEUS is an obstetrical curiosity remembered if only because of its strange name. There have been 180 cases reported in the literature, but so far as can be ascertained, none have been recorded in this country. Such fœtuses undoubtedly exist, as is evidenced by various museum specimens.

This case is reported because of the rarity of the condition and also the unusual presentation.

Clinical Record.

Mrs. Mc. was a *primipara*, aged twenty-three years, whose last menstrual period had occurred on July 9, 1951, and her expected date of confinement was April 20, 1952. The only abnormality during her pregnancy was an excessive increase in her weight at 22 weeks' gestation. This responded to the institution of a salt-free diet and rest. There was no history of vaginal bleeding during her pregnancy. On March 10, 1952, she presented with a story of "labour pains" since early morning, and about 8.30 a.m. she felt "something come away". There was a small "show", but no drainage of liquor.

FIGURE I.

Extreme flattening of the fœtus is well shown.

On examination of the patient, the *fundus uteri* was found to be at a level of thirty-five to thirty-six weeks' gestation, and contained a fœtus lying so as to present by the vertex in the left occipito-anterior position. The fœtal heart beat was regular and the sounds were clearly audible. In the vulva was a flattened, pale mass, which proved to be a *fœtus papyraceus*. This was suspended by a white cord approximately a quarter of an inch in diameter. The cord and the fœtus were completely avascular. The cord was divided and tied.

Throughout the remainder of the day (March 10) vague abdominal aching was present, but there were no true pains. As the *fœtus-in-utero* did not feel large, it was decided to let events run their natural course.

On March 11 irregular uterine contractions developed, associated with the passage of a watery, blood-stained discharge *per vaginam*. These contractions passed off until the afternoon of March 13, when they became of moderate nature. At this stage the fœtal heart became difficult to discern. The contractions again disappeared, and the fœtal heart became regular and audible. The patient had a restful night.

On March 14 moderate labour recommenced, and by 5.15 p.m. the fœtal head was at the level of the ischial spines and the cervix was almost fully dilated. A prominent bag of forewaters was present. Because of the prematurity of the fœtus and its previous distress, the decision was made to carry out a pudendal block and to perform a low forceps delivery. This was done after rupture of the membranes and the performance of an episiotomy. At 6.10 p.m. on March 14—that is, 106 hours after the birth of the *fœtus papyraceus*—a living female child was delivered. The third stage was normal. The placenta had two distinct sacs; one, presumably belonging to the abnormal fœtus, was small and that portion of the placenta was

pale and firm. A section taken between the two sacs revealed two amnions and no chorion—that is, the twins were uniovular.

The viable fœtus weighed five pounds three ounces and measured 19 inches in length.

The puerperium was complicated by separation of the *symphysis pubis*, which gave rise to considerable pain. This settled down with the application of a leather girdle.

The Specimen.

The overall length of the fœtus was six inches. It was very flattened and of white colour. No evidence of maceration was present—the scalp was covered with short, fine, dark hairs. The only abnormality was what appeared to be a complete absence of the auricle on the right side. This

FIGURE II.
Extreme flattening of the fœtus is well shown.

was in direct contrast to the left side of the skull, on which a well-formed auricle was present. Whether this was a congenital defect or due to pressure is not known. At one stage the fœtus was thought to be diencephalic, but dissection of the skull and X-ray examination proved only one set of bones to be present.

The age of the fœtus as determined by ossification shown by X rays was in the vicinity of twenty weeks. Its sex could not be determined. Examination of sections of the cord revealed no evidence of vascular anomalies.

Discussion.

The cause of fœtal death has been attributed to various factors—for example, placental degeneration, vascular anomalies in the placenta and cord—none of which offer a complete explanation. (F. J. Browne states that the communications between the circulations of uniovular twins are of importance from a pathological viewpoint, fœtal death *in utero* being three times more common in uniovular than in binovular twins.) Placental communications between the circulations of binovular twins are also said to exist.

The mechanism of fœtal death is that if one fœtal heart beats more strongly than the other, the circulation in the hypogastric arteries of the weaker will be reversed and its placental circulation stopped. In some cases, vaginal bleeding early in pregnancy may herald the blighting of one fœtus. In this case, the only abnormality during pregnancy was an excessive gain in weight at a period which was at the approximate time of fœtal death.

Death of one fœtus in a multiple pregnancy may have any of the following results: (*a*) expulsion of the dead fœtus with continuation of the pregnancy to term; (*b*) spontaneous abortion of both twins; (*c*) absorption of the dead fœtus if it is of less than two months' gestation (King); (*d*) retention of both living and dead fœtuses.

The fate of the retained dead fœtus may be one of three: (i) it may be macerated; (ii) it may become hard and fixed, but still retain its anatomical shape; (iii) absorption of *liquor amnii* and compression may occur, yielding a *fœtus papyraceus*. This is really an arrested process of maceration.

In most cases of *fœtus papyraceus*, the placenta is hard and pale, sections showing infarction and fibrosis. It may at an earlier stage show villous necrosis with masses of fibrinous material between the villi. This is the forerunner of extensive infarction and fibrosis, referred to above.

Perhaps in her wisdom Nature has destroyed an imperfect product, although there are no references to gross developmental abnormalities in reported cases. Whatever the cause of death may be, it is certain that the final papyrus-like form of the fœtus is due to mechanical compression, most probably by the head of its enlarging twin.

The cord is always thin and pale and may show vascular anomalies. In no case has a true knot of the cord been present.

There seems to be no difference in the incidence of *fœtus papyraceus* in monochorial or dichorial twin pregnancies, nor does the sex of the fœtus appear to play any part.

Kindred is of the opinion that "blighting of one twin may occur more often than should be expected in monochorial pregnancies of primiparous mothers". Death of fœtus is most likely to occur between the third and fifth months of gestation.

In most cases of *fœtus papyraceus* the viable child is born first, examination of placenta and membranes revealing the dead fœtus; but in several cases this has been reversed.

Kindred's review reveals that nineteen hours is the longest period between the birth of the dead fœtus and the viable child. In the reverse circumstances the longest recorded interval is three days.

O'Regan and Craig, however, report a case in which a *fœtus papyraceus* was expelled, and nine days later a normal child was delivered. On examination of the placenta and membranes, a second papyraceous fœtus was found.

Jerkuile and Parmelee report a pre-natal diagnosis of *fœtus papyraceus* when in an X-ray film taken at term they found, beside the bony structure of a full-term fœtus, that of a fœtus of four months' gestation, which proved to be a papyraceous fœtus.

Summary.

A case of *fœtus papyraceus* is presented, in which approximately one hundred and six hours elapsed between the birth of the subject and the birth of the viable twin.

A brief review of the literature is given.

Acknowledgements.

My thanks are due to the General Medical Superintendent of the Royal North Shore Hospital of Sydney, Dr. W. Freeborn, for permission to publish the case, and to Dr. S. Studdy, under whose care the mother was admitted to hospital. I should also like to thank Miss P. Simpson and Miss L. Mathieson for the preparation of photographs and skiagrams.

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EXCISION OF URETHRAL STRICTURE: FOLLOW-UP AFTER FOURTEEN YEARS.

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THE patient, W.D.G., a male, aged fifty-three years, who in his earlier years had been a sailor, was first examined in 1939, when he complained of difficulty in passing urine during the preceding three months. He had first had urinary symptoms in 1915, when aged twenty-eight years; a urethral stricture of undetermined ætiology was found and dilated. Intermittent dilatations had been carried out at varying intervals, the last dilatation being fifteen months prior to his first being examined by us. He had had an episode of acute retention of urine in 1935, relieved by dilatation. Ætiological factors (urethritis and trauma) were denied.

On examination of the patient, a hard, fibro-cartilaginous mass was palpated in the perineum in the region of the bulbous urethra, and examination *per rectum* showed the prostate to be of normal consistency and not enlarged. The blood urea content was estimated at 34 milligrammes *per centum*. A urethrogram (Figure I) showed a urethral stricture.

Urethral dilatation was attempted on three occasions and there was no passage even to filiform bougies, so a suprapubic cystostomy was performed on October 30, 1939.

On December 4, 1939, as there was still no success in passing urethral instruments, excision of the urethral stricture was performed, as is strongly recommended by Hamilton Russell (1915) in such cases.

An inverted Y-shaped incision was made in the perineum and the bulbous urethra and the surrounding dense fibrous tissues were incised longitudinally. A Syme's staff was next passed down the urethra to the stricture, the urethra was mobilized on either side of the stricture and the affected part of the urethra was excised. The tissue removed was reported on by the pathologist (Dr. G. F. Davies) as "urethral stricture: chronic inflammation". The mobilized divided ends of the urethra were drawn together and sutured around their whole circumference without a urethral catheter, and the perineal wound was closed with interrupted sutures of black silk in the skin.

The actual technical steps of the operation are exactly those of the operation described in Thomson Walker's "Genito-Urinary Surgery" (1950), in that the whole circumference of the urethra was sutured and the urinary stream was diverted suprapubically. (In the Hamilton Russell operation the urethral ends are united as a ribbon and reliance is placed on perineal urethrostomy for urinary diversion.)

On January 25, 1940, the patient was anæsthetized and the urethra was found to admit Lister's curved metal bougies to size 15/18 English (30 French) easily. A urethrogram made with sodium iodide solution (5%) showed a normal urethra at this time (Figure II).

The patient was discharged from hospital on February 5, 1940, with the suprapubic fistula closed and a normal urinary stream.

He was readmitted to hospital on February 16, 1940, with intense frequency of micturition, scalding, profuse hæmaturia (mainly terminal) and pain, and extreme tenderness above the inguinal ligaments and in the adductor muscles. Microscopic examination of the urine revealed "pus 10%" and on culture *Bacterium coli* grew profusely. The urinary symptoms were regarded as being due to acute cystitis and responded to alkalinization of the urine. The extreme pain in the adductors (preventing walking), which may well have been due to *osteitis pubis*, subsided with physical therapy, so that on March 19, 1940, the patient was discharged from hospital with slight adductor tenderness only and an "improving gait".

The next passage of urethral bougies was two months later (May, 1940), and instruments were passed in 1946 and again in 1948.

In January, 1952, he reported again, complaining of frequency of micturition and diminished force in the urinary stream. A urethrogram was made, "Diodone" (35%) being used; this revealed a moderate recurrence of the urethral stricture (Figure III). It is interesting to note that in this picture the prostatic urethra is now somewhat elongated and there is some intravesical bulge, demonstrating some benign prostatic hypertrophy. An excretion urogram reveals a normal upper portion of the urinary tract and bladder shadow. Normal renal function was further confirmed by a blood urea reading of 30 milligrammes *per centum*, the urinary urea content reaching 2·25 grammes *per centum* (third hour) in a urea concentration test. The urine contained pus cells $> 20 < 60$ per high-power field, and clumps (centrifuged specimen). *B. coli* was grown on culture.

Urethral dilatation to 13/16 English (27 French) was performed, and examination showed that the patient had no residual urine. He was again discharged from hospital with a good urinary stream.

He reported again on March 17, 1953, and his urethra was dilated to 15/18 English (30 French).

In retrospect, it is considered that the operation of excision of the urethral stricture has been of value in this case and extended the intervals between which dilatation has been necessary.

Discussion.

The older and still the basic treatment of urethral stricture is intermittent urethral dilatation, which, according to Attwater (1943) has been practised since the sixth century B.C., when the Hindoos treated urethral strictures with graduated dilators of metal or wood. Attwater states that through the centuries various other forms of treatment have been described, such as the operation of *la boutonnière* (a buttonhole puncture of the dilated urethra behind a stricture), as used by Chopart (1786); Baron Dupuytren's method of "vital dilatation", an indwelling catheter or bougie being used; Maisonneuvre's operation of internal urethrotomy (1848); and the Wheelhouse method of external urethrotomy (1876).

All these methods produce improvement, and after such operations as internal and external urethrotomy intermittent urethral dilatation must be continued. A desire to produce more lasting improvement has led to attempts actually to cure the lesion by excising it and reuniting the healthy ends of the urethra (Hamilton Russell, 1915; Thomson Walker, 1950), or by reconstructing a part of the urethra by some plastic procedure, as, for example, along the lines of the Ombrédanne operation (Walters, 1935).

Hamilton Russell was an advocate of stricture excision and reported (1915) eight successful cases. The features of the Russell operation were (*a*) an incision as for perineal prostatectomy, (*b*) a median longitudinal incision over the urethral stricture, (*c*) excision of the stricture with suture of the roof of the urethra only, no indwelling catheter, (*d*) a perineal urethrostomy to divert the urine.

Even after excision of a urethral stricture bougies should be passed at regular intervals to maintain the patency of the urethra. Good results are obtainable in selected cases.

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SIDELIGHTS ON CHLOROQUINE: TWO CASES OF INTEREST.

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THIS paper deals with an attack of *vivax* malarial fever in a breast-fed infant whose mother was taking chloroquine as a malaria prophylactic, and an amœbic abscess of the liver in a man who was taking chloroquine as a malaria prophylactic.

Case I.

Anthony H., male, white, then aged four weeks, was first seen on May 26, 1952, suffering from diarrhœa with occasional greenish stools; he recovered promptly under treatment. On June 16 he had a seborrhœic rash on the eyebrows and face, which responded promptly to treatment. These were his only illnesses since birth.

On June 1, 1952, his mother, who had been taking 100 milligrammes of proguanil hydrochloride (as "Paludrine") daily as a malaria prophylactic, was changed over to 250 milligrammes of chloroquine diphosphate (as "Aralen") three times a week, in the course of a controlled experiment in malaria prevention. Anthony, who was still being breast-fed, was given no direct prophylaxis.

On September 19, now aged four and a half months, he developed a temperature of 101° F. at 6 p.m. He had been fretful all afternoon, refusing all food. During the night he had a small sweat and he seemed well the next day; a blood film taken then showed no malaria parasites. But on September 21 he had another afternoon rise of temperature, with recurrence of the other symptoms. A diagnosis of clinical malarial fever was therefore made, and his mother was given five grains of quinine bihydrochloride six times a day. Anthony immediately began to object to his mother's milk, but took it under protest when he became hungry.

The mother, however, was able to take only five doses of quinine, after which she became giddy and vomited every dose (she had had trouble in taking quinine some six years previously, during induction of labour). She was therefore given 250 milligrammes of chloroquine every day. Anthony appeared to be well thereafter and had no rise of temperature on September 23 or 25; but on September 27 his temperature rose to 102° F. and a blood film showed *Plasmodium vivax* in moderate numbers. He was then given one grain of quinine bihydrochloride twice daily by mouth; he vomited the first dose, but thereafter took it well. He had another small rise of temperature on September 29, and a film taken next day showed a few *vivax* parasites. From then on he was very well, and a film taken on October 3 showed no parasites; a few of the polymorphonuclear leucocytes contained black pigment granules. In all, he had 10 grains of quinine over seven days. He has been well since. His mother has been changed back to 100 milligrammes of proguanil daily, and he is receiving no direct prophylaxis. Ultimately, when he is weaned, both he and his mother will go on to chloroquine prophylaxis.

Case II.

J.M., male, white, aged fifty-four years, has no history of any form of dysentery at any time. On May 17, 1952, he reported that he was always weary, was not eating or sleeping well, and occasionally vomited after a meal. He admitted that he had been taking far less than the routine dosage of proguanil (100 milligrammes daily is recommended here). His temperature and pulse rate were normal; he had no abnormal signs in his abdomen; a blood film showed no malaria parasites; his hæmoglobin value was 70%; he had not been losing weight; and no other abnormality was found. It was decided that he had probably been suffering from incompletely suppressed malaria and he was given 200 milligrammes of camoquin hydrochloride three times a day for five days, and thereafter 250 milligrammes of chloroquine diphosphate (as "Aralen") three times a week. He was also given iron and liver, and a bismuth mixture. By May 21 he was feeling much better; on June 21 his hæmoglobin value was 80% and he needed no further treatment beyond a continuation of the iron and liver.

On September 25, 1952, he complained of epigastric pain during the previous two days, with occasional vomiting. On examination he showed some epigastric tenderness; his temperature and pulse rate were normal; there were no malaria parasites in a blood film. He claimed to have taken all chloroquine as ordered. The next day he complained of pain in the region of his xiphisternum, radiating laterally across his lower ribs; both *rectus abdominis* muscles were tender as far down as the umbilicus. His temperature was 100·6° F. and pulse rate 94 per minute. A diagnosis of Bornholm disease was made (an epidemic was in progress at the time) and a mild sedative was prescribed.

When seen again on September 29 he was complaining of very acute subxiphisternal pain; his epigastrium felt full and tense, and his liver margin was tender; his tongue was heavily coated and he was constipated; his evening temperature was 101·6° F. and his pulse rate was 100 per minute. The previous diagnosis of Bornholm disease had obviously to be abandoned, and on the following day, with his white cells 14,650 per cubic millimetre, temperature 102·2° F., and pulse rate 98 per minute, the correct diagnosis of amœbic abscess of the liver was arrived at. Intramuscular injection of emetine hydrochloride, one grain daily, was immediately started, with the usual result—by the fourth day of treatment his temperature had fallen by slow lysis to almost normal, the subxiphisternal and epigastric pain and tenderness had faded to a vague "heavy feeling", his tongue was clean, and he was beginning to eat. Ten days after that he returned to light work, feeling a little shaky but quite well. He will receive follow-up courses of emetine and quinoxyl in a month's time, and he is continuing to take chloroquine in normal doses for malaria prophylaxis.

Discussion.

It is known that chloroquine is concentrated in the liver; and this is not the case with the other antimalarial drugs. When a nursing mother is given adequate doses of quinine or "Atebrin", an adequate proportion is secreted in her milk and received by the infant. I have always assumed that the same applies to proguanil, and during almost five years' experience with this drug I have seen nothing which suggests the contrary. Now Anthony is one of the only two breast-fed infants whose mothers have been taking chloroquine under my supervision, and he developed malarial fever after three and a half months. The other infant has not shown any signs of infection during the same period, but the other factors are too variable for this to mean anything. In any case, the mother of the second infant has now gone back to proguanil at her own insistence, so no further information can be expected. However, in Anthony's case the most likely explanation is that so much of the chloroquine was taken up by his mother's liver that he did not receive a protective amount in her milk.

On the other hand, it is obvious that however much chloroquine was concentrated in J.M.'s liver, it was not enough to prevent an amœbic abscess forming, or, if his vague upset four months previously is to be regarded as the first evidence of amœbic hepatitis, the chloroquine which he was taking can be regarded as only retarding, but not preventing, the development of an abscess, and as tending to obscure the diagnosis.

Summary.

Considering these two cases, it is suggested that, when normal suppressive doses of chloroquine are being taken, the concentration of chloroquine in the liver is great enough to prevent a malaria-suppressive amount from being excreted in the milk of a nursing mother, but not great enough to protect a man from developing an amœbic abscess of the liver.

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THE DIAGNOSIS OF CANCER OF THE STOMACH.

ALTHOUGH the problem of diagnosis of cancer of the stomach has not been discussed in these columns for several years, the problem is still as acute as it ever was. Physicians and surgeons and general practitioners will all agree that any measures which are likely to facilitate early diagnosis should be studied with care. Some years ago, we quoted in this journal the statement by Frederick Christopher, and attributed to Collins, to the effect that diagnosis of cancer of the stomach should be made when the patient appeared normal, when there was no palpable tumour, when there was no reduction in the erythrocyte count and hæmoglobin value, and before loss of weight had occurred. Obviously this counsel of perfection is seldom, if ever, attained. It is commonly accepted that a patient who is suffering from a gastric ulcer should, in ordinary circumstances, seek treatment at the hands of an experienced physician, firstly because this is the safest course to adopt, and secondly because it is often completely successful. In regard to this, we have previously recalled Moynihan's declaration that success of the medical treatment in early cases of carcinoma of the stomach was one of the causes of the high mortality of the disease. One of the unfortunate factors is that patients suffering from gastric symptoms are, more often than not, slow to seek the advice of a medical practitioner. In a recent contribution, William A. Cooper[1] has stated that this is founded on wilful neglect, but has its origin in ignorance, fear, and the hope that next day or week will be better. His report deals with 687 cases of gastric cancer, and he found that delay on the part of the patient in this group had been about eight months. It might also be noted that, on an average, it took the medical attendant four months after he first saw the patient before he arrived at a definite diagnosis. This means that these 687 patients, on an average, harboured a gastric cancer before the definite diagnosis was made and treatment was attempted. With no wish to labour the subject, we may repeat a statement made in May, 1948, that at that time the number of deaths in Australia attributed to cancer of the stomach and duodenum every year was between 1750 and 1830. Cancer of the stomach and duodenum was thought to be the cause of 20% to 22% of the total deaths attributed to cancer among the Australian population, and about 26% of these deaths occurred among the male population.

Of the aids to diagnosis in cancer of the stomach, radiological examination is still the most useful method. Cooper points out that it is only as valuable as the experience of the radiologist is great, and that in the most experienced hands it has many pitfalls. Some years ago we quoted a statement from the Mayo Clinic that more than half the gastric growths which were found to be inoperable had not been diagnosed as inoperable by radiological examination; the series on which this observation was made numbered 400. It may quite well be that an improvement on these figures has by now been effected. Cooper sums up the radiological position as follows: (i) In the hands of experienced radiologists, the method is extremely accurate in the detection of pathological conditions in the antrum and along the lesser curvature of the stomach. (ii) This accuracy decreases in the upper one-third of the lesser curvature, and in the anterior and posterior walls and greater curvature. (iii) In the cardia, the method is very inadequate, being subject to error in about half the cases. (iv) In the naming of pathological conditions, the experienced radiologist, under ideal circumstances, has an inherent error of 10% to 15%. In less experienced hands, the accuracy of the method decreases sharply in all the categories mentioned. Our object on the present occasion is not to discuss the whole range of diagnostic methods; no reference will therefore be made to the gastric acid level, the presence of occult blood in the stools, or gastroscopy. Our object is to draw attention to Cooper's views on the cytological examination of the gastric sediment. In 1950, W. A. Cooper with F. G. Panico and G. M. Papanicolaou described an "abrasive balloon" which was used for exfoliation of gastric cancer cells.[1] It will be remembered, of course, that Papanicolaou has done a great deal of work on the diagnosis of uterine cancer by cytological methods. In 1950, the three authors recorded the study of 70 patients on whom the balloon was used. They reported confirmation of the diagnosis in 33 cases. They explained that mechanical irritation of the gastric mucosa caused increased exfoliation of the abnormal proliferative gastric cells, and they reproduced photomicrographs of well-preserved normal gastric cells and of well-preserved gastric cancer cells. In his recent article, Cooper refers to the previous study reported in 1950. He states that in the 1950 study, the balloon had been used only in cases in which the diagnosis was known. He deals in his 1952 study with the first 200 cases in which an attempt has been made to evaluate the work from the clinical standpoint. The 200 patients collected suffered from pathological gastric conditions, and were to be treated surgically. Most of the cases presented diagnostic problems, and the cytologist knew nothing of the clinical picture at the time the smear was examined. In the 200 cases, there were 45 of malignant gastric disease and 155 of non-malignant gastric disease. In the 45 cases in which the final diagnosis was malignant gastric disease, five of the cytological readings

[1] *J.A.M.A.*, October 18, 1952.

[1] *J.A.M.A.*, August 12, 1950.

were negative, seven were suspect, and 33 were positive; errors were five in number. In the 155 cases in which the final diagnosis was non-malignant gastric disease, the cytological readings were negative in 146, suspect in 8, and positive in 1, the error thus being one. Of the five malignant gastric lesions which were missed, one was a lymphosarcoma and the other four were gastric cancers. One of these was a narrow, distal antral lesion which probably was not reached by the balloon, and the other three were ulcerating lesions covered by a necrotic membrane in which no viable tumour cells reached the surface of the gastric mucosa. The cause of the error for the one false positive report was "not clear".

The full details of the procedure need not be given in this place. It must suffice to point out that the balloon, which is covered with a fine netting, is passed into the stomach. When it is inflated with 175 cubic centimetres of air, the balloon measures 10 by 5 centimetres. The tube on which the balloon is placed has a double lumen. One lumen is fitted to a syringe and the stomach is aspirated, the material being used for the fasting specimen of a gastric analysis. After histamine has been given, and several specimens have been collected for gastric analysis, the balloon is inflated with about 150 cubic centimetres of air, and the tube is withdrawn until the balloon is checked at the cardia. Peristaltic action carries the balloon down to the pylorus in five to ten minutes. The balloon is drawn back to the cardia again, and the process is repeated five to six times. After the balloon has been deflated and withdrawn, slides are prepared from the material on the netting. For preparation of the slides, the material is washed off in Ringer's solution mixed with 95% alcohol, and after centrifugalization, the sediment is stained according to the Papanicolaou technique. The apparatus has not been used in the investigation of any patient who has suffered from recent hæmorrhages, or who is known to have œsophageal varices. We are told that the aspirated material is frequently tinged with blood, particularly if a lesion is present, but no hæmorrhage has occurred in over 200 trials with the method.

Cooper writes that among the lesions correctly diagnosed by his smear technique, were four that had the gross appearance of benign gastric ulcers. In one of these cases, the malignant area was not more than five millimetres in diameter. He claims for the method great accuracy in the differentiation of benign from malignant ulcers. In the first 100 cases four of the cancers were diagnosed as benign by radiological examination, and eleven of the benign lesions were thought to be cancerous—fifteen misses in all. In only one of the fifteen cases was radiological examination thought to reveal no abnormality. Cooper concludes that the method of examination with the balloon technique provides another approach to the diagnosis of gastric cancer which is at least as accurate as radiological examination. We must agree with him that the method has inherent accuracy that is lacking in X-ray diagnosis, in that it reveals the basic pathological nature of the growth. He is wise enough to state that the final evaluation of the method will have to await confirmation through use by other workers. He draws attention to the fact that very little is known of the value of the method in early gastric cancer, since so few small lesions are seen. So far as can be discovered, this method has not yet been used in Australia. It seems probable that the method will provide an additional means of diagnosis. We may be quite certain that no method of investigation will produce reliable findings in every case, but on the face of it, an attempt should be made to discover the real place of Cooper's method in the field of diagnosis. Every additional and reliable means of investigation, though it will not always be successful, will surely help to reduce the four months period mentioned by Cooper as having elapsed between the first examination of a patient by the medical attendant and the making of a final diagnosis. There is no doubt also that promptness and accuracy of diagnosis will help to reduce the other lamentable period of eight months which Cooper found to occur between the first appearance of symptoms and the patient's first visit to his doctor.

Current Comment.

DIABETIC GLOMERULOSCLEROSIS.

In 1936 Kimmelstein and Wilson described glomerular lesions in the kidneys of diabetics, and the associated vascular lesions have been recognized since as being of importance in the subjects of this disease. The name glomerulosclerosis has been used both with and without qualification, such as "intercapillary", but G. F. M. Hall, who has published a study of the condition based on post-mortem material, considers that the qualifying term, although used by the original workers, has not been proven to be quite correct.[1] Hall presents a review of the literature, with a summing-up of his own analysis of autopsies of 120 subjects of diabetes, and as a control has also examined a series of 100 non-diabetic subjects who came to autopsy from other causes. The criterion can easily be established, as the glomerular lesions are characteristic, being nodular, with hyalinization of the adjacent arteriole, and in severer cases, a great multiplication of the glomerular nodules, secondary atheromatous narrowing of the renal arteries, and terminal ischæmic obliteration of the glomeruli. These changes are well shown in accompanying sections. Using these criteria, Hall has divided his material into grades of severity, calling them mild or severe, and finds that there is a definite correlation between severity of the lesions and duration of the disease, and this relation is statistically significant. The percentage of incidence rises from 26 in disease of five years' standing to 63 in a duration of sixteen or more years. Severity and the degree of control are harder to estimate, but the amount of insulin needed to effect control was taken as a guide; severe grades of the disease were taken to be those which required 30 or more units of insulin a day. Here, too, there was a definite degree of correlation, not so much in the existence of glomerular change, but in its severity. Certain other factors were considered, but were not thought to be relevant. One of these factors was the presence of amyloid disease of the kidney, which has been mentioned in the literature by reason of the superficial resemblance of some of the anatomical lesions to amyloidosis. No support was found for this suggestion. Cirrhosis of the liver has also been thought to have some connexion with the occurrence of glomerulosclerosis, but the author can find no ætiological connexion between the two conditions. After a general discussion of the available material in the literature and in this research, Hall concludes that the total incidence in diabetics of this glomerular nodular affection is 37%, and that it does not occur in non-diabetic persons. It is rather commoner in women than in men, but the relationship to advancing age is more definite. Hall thinks that poor control of diabetes is an important

[1] *Quart. J. Med.*, October, 1952.

factor in its ætiology, though the condition occurs in both mild and severe diabetes.

This work helps to give a clearer concept of the ætiology of this apparently specific part of the diabetic complex. Its later relationship with renal and general arterial sclerosis of the familiar pattern is less clear, but Hall has studied this aspect of the syndrome and concludes that, while hypertension is associated frequently with diabetic glomerulosclerosis, it cannot be regarded as an ætiological factor. Nevertheless, this does not raise any objection to considering diabetic glomerulosclerosis as one manifestation of generalized vascular degeneration in diabetes. Death may occur from renal failure, due apparently to the later lesions in the renal and arteriolar tissues, and in this event severe atheromatous constriction of the renal arteries has been observed. Hall remarks that the glomerular lesions, with their terminal relation to renal atheroma, hypertension and arteriolar sclerosis and retinal microaneurysms, may be regarded as different manifestations of generalized vascular degeneration in diabetics. Once again, it would seem that the study of morbid anatomy has something to teach us in the complicated subject of vascular disease.

RHEUMATIC FEVER.

A SYMPOSIUM on rheumatic fever was held at the annual session of the American College of Physicians in 1952. Some of the papers have been published[1] and give an extremely informative review of the disease and of modern methods of prophylaxis. Even though rheumatic fever is on the decline and is apparently less severe nowadays, it still accounts for all too many deaths and a great deal of disability in the prime of life. A most forceful reminder of this and of the need to do all that is possible to prevent the disease is provided by the account by E. F. Bland and T. D. Jones of the natural history of rheumatic fever. They begin by describing the major and minor manifestations of the initial illness—carditis (in 65% of cases), arthralgia (41%), arthritis (40%), chorea (51%), subcutaneous nodules (10%), nosebleeds (27%), fever, rashes, abdominal and precordial pain, pulmonary changes—giving a particularly good *résumé* of the early signs of valvular involvement. They then go on to give the results of a twenty-years follow-up study of 1000 cases. The subjects were young patients with an average age of eight years at the first attack. Two-thirds of them developed rheumatic heart disease, and one-third (301) died in the twenty-years period; 20% (202) had died within ten years. The most serious prognostic features during the initial illness were a greatly enlarged heart (80% of these patients died within ten years), congestive failure (71%) and pericarditis (56%). Less ominous signs were nodules (associated with 38% of deaths in ten years) and arthritis (22%); while chorea was associated with a relatively benign form of disease, as only 9% of the 518 patients with chorea succumbed within ten years. Signs of well-defined valvular disease disappeared entirely in 11% of cases in ten years and in 16% in twenty years. On the other hand valvular disease, most often "pure" mitral stenosis, developed in 44% of patients who showed no signs of carditis during their initial illness. Chorea was a prominent feature in this group, and in only one-third could the development of carditis be attributed to recurrent attacks of rheumatism. Thus, at the end of twenty years, when the average age would be twenty-eight years, 301 of the 1000 patients had died, and 699 were living. Of the survivors, 380 had valvular lesions.

There is obviously no room for complacency in treating this disease, and the most effective treatment is prevention. In another article of this symposium H. B. Houser and G. C. Eckhardt describe the recent developments in this field. First, they stress the importance of dealing effectively with the streptococcal infection which usually precedes the onset of rheumatic fever. Their own results substantiate the claims made by other writers: 23 cases of rheumatic fever followed streptococcal infection in 1142 control patients, whereas only two cases occurred in 1120 patients who were treated with penicillin. Similar results were obtained with aureomycin. Secondly, they emphasize the value of recognizing and treating promptly any streptococcal tonsillitis or pharyngitis occurring in patients with a history of rheumatic fever. But in 40% of instances these infections are too slight to cause medical help to be sought, and therefore the third aspect of prophylaxis against recurrent attacks is all-important—the daily administration of sulphadiazine or penicillin. The value of this has been adequately proved, and the dangers of toxic reactions and drug-resistant bacteria are slight. A useful scheme of treatment is to be found in the American Heart Association's statement[1] to which we referred in detail in these columns on March 28, 1953. This statement recommends that prophylactic treatment should be started in the third week of the attack of rheumatic fever and continued up to the age of eighteen years or for at least five years in older patients. The dosage is 0·5 to 1·0 gramme of sulphadiazine given daily or 250,000 units of penicillin by mouth twice a day. The statement also includes points of diagnosis and treatment of acute streptococcal throat infections.

The cause of rheumatic fever remains unknown, but streptococcal infection plays an important part. In the excitement of following the advances being made by cardiac surgery in bringing temporary relief to a few surviving sufferers of the disease we should not forget the proverbial stitch in time, and remember to strike the streptococcus.

[1] *Ann. Int. Med.*, November, 1952.

SMOKING AND VITAMIN C.

THE famous "Counterblaste to Tobacco", written by King James I, was at first published anonymously in 1604. This verbal attack was followed by enactments prohibiting the importation of tobacco in any form, but the attractions of the Indian weed proved too powerful even for the divinity which hedged the King of England and Scotland. In Victorian times smoking was somewhat under disapprobation and was never indulged in when members of the fair sex of any social standing were present. But the victory of tobacco in the present century has been complete and the cigarette has broken down all inhibiting barriers; incidentally it has induced among many of its addicts a curious lack of good manners. In the last few years we have heard warnings concerning the possibility of cancer of the respiratory passages being induced by smoking; some medical publicists are dogmatic in their condemnation, whilst others adopt a more critical and cautious attitude. The public, however, has not taken such warnings to heart, and so the tobacco manufacturer and retailer continue to live in comfort, whereas the grower continues to work hard and earn little.

The latest attack on smoking emanates from the medical school of the city of Lodz. Two Polish investigators, F. Venulet and Z. Moskwa,[2] announce that tobacco smoke produces a partial disintegration of vitamin *C*. Those who smoke and those who breathe air contaminated with tobacco smoke and those who work in tobacco factories show a marked diminution of vitamin *C* in urine, blood and internal organs; but a return to normal follows abstention. The high content of vitamin *C* in pituitary, adrenal cortex and other endocrine glands indicates that this vitamin is carrying out important duties in these essential organs. The well-known poisonous action of tobacco on the heart can be explained, at least in part, by the same destructive action. The widespread prohibition of smoking in pulmonary tuberculosis and peptic ulcer finds here an explanation. These Polish investigators claim that women are more prone than men to the evil effects of vitamin *C* reduction, but just why they do not explain. Confirmation or the reverse of these conclusions will be awaited with interest.

[1] *Lancet*, February 7, 1953.

[2] Venulet, F., and Moskwa, Z. (1952), *"Influenza del fumo di tabacco sulla vitamina C contenuta nell'organismo"*, *Omnia Medica* (Pisa), 30: 279.

Abstracts from Medical Literature.

GYNÆCOLOGY AND OBSTETRICS.

Carcinoma of the Vulva.

J. W. NEWELL AND D. G. MCKAY (*West. J. Surg.*, August, 1952) present a series of 53 cases of carcinoma of the vulva from the Free Hospital for Women, Boston. The incidence of the disease was 1·4% of primary malignant tumours at the hospital; the average age of the patients at the time of the first visit was 61·6 years, and 16 patients were *nulliparæ*. Without exception, the patients complained of a localized mass, painful ulcer, discharge, vulval irritation or bleeding, the symptoms occurring singly or in various combinations. Pain was usually an early symptom; of patients with lymph node involvement, two-thirds had had symptoms for less than one year. There was no correlation between tumour size and node involvement. The majority of the tumours arose in the *labia majora*, and most were in the anterior half of the vulva. Six of the 53 patients had another primary carcinoma, two being in the breast, one squamous carcinoma in the cervix and one adenocarcinoma in each of the stomach, endometrium and sigmoid colon. In the series no truly undifferentiated tumours were seen on histological examination, and there was no correlation between histological grade and presence or absence of lymph node involvement. The authors state that the prevention of carcinoma of the vulva at the present time consists in recognition and adequate treatment of all leucokraurotic lesions, which in turn will provide adequate control in about four-fifths of those women destined to develop this disease. No therapy other than surgery will cure leucokraurosis, although spontaneous cures may occur. Once carcinoma appears, the authors recommend a broad and radical surgical attack, irrespective of the size, duration of symptoms, location or histological grade. Of 12 patients treated by radical vulvectomy and bilateral radical groin resection, eight were alive and free of disease at the end of five years.

Carcinoma of the Uterine Cervix.

J. W. KELSO (*Am. J. Obst. & Gynec.*, May, 1952) reports the results of surgical treatment of 100 patients with carcinoma of the *cervix uteri*; 92 had the Wertheim radical operation, five had additional surgery in the form of cystectomy with ureteral transplantation (two cases) and abdomino-perineal resection (three cases), and three were eliminated from the analysis for various reasons. These patients represent all who came under the author's care during the period 1945 to 1951 with cancer of the cervix. The condition was considered to be invasive carcinoma in all cases. Patients were grouped clinically according to the Schmitz classification as follows: group I, 15 cases; group II, 47 cases; group III, 30 cases; group IV, five cases. Histologically, 82% of the tumours were epidermoid carcinomata, and 9·7% were columnar-cell carcinomata. Routine pre-operative treatment included restoration of the blood to a normal level of hæmoglobin, preparation of the bowel with "Sulphathalidine", penicillin cover, and an injection of lipo-cortical hormone on the morning of operation. Cystoscopic examination and catheterization of the ureters were performed immediately before the operation, and the anæsthesia of choice was spinal anæsthesia supplemented by intravenous administration of "Pentothal Sodium". The first 50 patients had preliminary treatment with radium (3500 milligramme hours), but this technique was abandoned in the remaining 50 cases. The technique of the operation combines the Wertheim hysterectomy with transperitoneal lymphadenectomy. Operative mishaps have included severing a ureter, cutting the right common iliac artery and injury to branches of the sciatic nerve. The author considers that the control of hæmorrhage in the obturator region is the chief technical difficulty. Routine post-operative care includes adequate sedation, intravenous fluid therapy, and penicillin and lipo-cortical hormone injections. The outstanding post-operative complications were those of the lower part of the urinary tract; six uretero-vaginal fistulæ and two vesico-vaginal fistulæ followed the 92 Wertheim operations. There were two deaths due to operation in the 95 patients submitted to radical surgery. The author considers that radical surgery according to this technique has a place in the treatment of carcinoma of the cervix and the operative mortality rate should be very low.

Stress Incontinence of Urine.

ALBERT ALDRIDGE (*J. Obst. & Gynæc. Brit. Emp.*, October, 1952) discusses the control of micturition and the causation and treatment of stress incontinence, with particular reference to his own operation in which an aponeurotic fascial sling is used for the support of the proximal end of the urethra. He states that stress incontinence usually follows obstetrical trauma, surgical trauma or unsatisfactory wound-healing following vaginal plastic operations. Predisposing factors in its development are congenital under-development of the urethra and its supports and involutionary changes incidental to the menopause. The control of urination is said to depend on normal functional balance between the urethral sphincter mechanism and the expulsive force of the bladder. Conditions which may cause abnormally high intravesical pressures are pelvic tumours causing external pressure on the bladder, uterine prolapse, and abnormal innervation, as with *spina bifida*, spinal injuries or multiple sclerosis. The author stresses the importance of determining whether the leakage of urine is due to urge incontinence or stress incontinence. As a rule stress incontinence is associated with displacement of the urethra and bladder neck in a downward and forward position beneath the pubic arch. The calibre of the urethral lumen is increased and its length may be reduced because its proximal third remains open and becomes an extension of the bladder. As yet, knowledge is limited of the mechanism by which urination is controlled. This limited knowledge and clinical experience indicate three principles in the treatment of stress incontinence: reduction of the lumen of the over-stretched urethra to normal size and restoration of the length of the shortened urethra; repair of the damaged, over-stretched and retracted urethral sphincter muscles; construction of a support for the urethra and bladder neck. X-ray studies by Jeffcoate suggest that urinary control depends on the maintenance of a proper angle between the proximal end of the urethra and the base of the bladder. The author states that operations for the relief of stress incontinence should not be undertaken until six months after the original injury or operation for the cure of the condition. He considers that 80% of women with stress incontinence can be cured by vaginal plastic operation; 10% to 20% of women remain unrelieved, and suprapubic operations, either alone or in combination with vaginal plastic operations, have been employed in these cases. The author has devised a fascial support for the relief of stress incontinence in such cases and considers it to be a simple and safe procedure. The sling operation may fail if the fascial support is not made sufficiently taut, if the abdominal wall is too relaxed, or if the urethra and vesical neck cannot be freely mobilized on account of scar tissue. The author considers that it will be difficult to develop a well-defined operation for stress incontinence until exact knowledge is available concerning the control of urination. At present, vaginal plastic surgery should be the choice for a high percentage of women who have primary stress incontinence. A suprapubic operation should be considered, according to the author, for women who have had one or more failures with vaginal plastic surgery.

Cancer of the Body of the Uterus.

M. A. VAN BOUWDIJK BASTIAANSE (*J. Obst. & Gynæc. Brit. Emp.*, October, 1952) reports a study of 264 patients with cancer of the body of the uterus; 217 underwent vaginal hysterectomy. At the Women's Clinic of the University of Amsterdam the author found an incidence of one case of carcinoma of the body of the uterus to 2·8 cases of cervical carcinoma. Of the 264 patients with *carcinoma corporis uteri*, 51% continued to menstruate until after they were fifty-two years of age. The frequent occurrence of a late menopause and the association of hyperplasia with carcinoma of the corpus suggest that œstrogenic substances are of ætiological importance. The author found endometrial hyperplasia together with carcinoma in 21 cases. He considers that carcinoma particularly occurs as a transformation of atypical hyperplasia. The highest incidence of corporeal carcinoma in this series of cases was in the age group fifty-one to sixty years. Although vaginal smear examination frequently arouses the first suspicion of carcinoma, a positive diagnosis can be made only on histological examination of endometrium removed by curettage. The author considers that functional uterine bleeding in women over fifty years of age carries an increased risk of cancer, and if hormonal treatment fails, treatment by hysterectomy is preferable to treatment by X-ray castration. He states that there is almost universal agreement that operative treatment is preferable to radium therapy for cancer of the body of the uterus; some authorities prefer a pre-operative course of radium. He considers the risks of implantation of cancer cells at operation to be much less after vaginal hysterectomy than after abdominal hysterectomy. Only one vaginal vault recurrence followed

217 vaginal hysterectomies, but three vault recurrences followed 33 abdominal hysterectomies. Radium therapy prior to vaginal hysterectomy is not recommended by the author. It causes delay in radical surgery, latent infection may be activated, and cancer cells may be freed by manipulation. An alleged disadvantage of vaginal hysterectomy is that removal of the adnexa is not always possible in operations by this route. The incidence of ovarian metastases in body cancer has been reported at 14 cases in 204 hysterectomies, but the author considers that when spread of cancer has involved the ovaries, other parts of the body may be already involved. The author's five-year survival rates were: 75·3% when both appendages were removed; 50% when the appendage on one side was removed; 70% when neither ovary was removed. He considers that the operability rate is higher with vaginal hysterectomy than with abdominal hysterectomy. Thirty-three patients in this series had abdominal hysterectomy chiefly because the uterus was too large to remove by the vaginal route. The operative mortality rate in 217 patients treated for cancer of the body of the uterus by vaginal hysterectomy was nil. The author compares results according to the type of surgery used as follows: vaginal hysterectomy—five-year cure rate 72·2%, ten-year cure rate 70·2%; abdominal hysterectomy—five-year cure rate 58·3%, ten-year cure rate 57%. The author observes that the mortality rate increases greatly with the age of the patients and the results of both operations are much worse in patients over fifty years of age.

Pregnancy and Congenital Polycystic Kidney Disease.

NORMAN MORRIS (*J. Obst. & Gynæc. Brit. Emp.*, December, 1952) describes four cases of polycystic disease of the kidneys complicating pregnancy and two additional cases in which the disease was first diagnosed several years after the last pregnancy. He suggests that as relatively few other cases have been reported, either the diagnosis is frequently missed or fertility is seriously affected. The adult form of the disease is inherited as a Mendelian dominant; and if a woman is affected, there is a 50% chance that her offspring will also have the disease. The youngest age at which the adult form of the disease was detected was eleven years—the "infantile" type appears to be of different ætiology—hence there appears no justification for terminating or forbidding pregnancy on the grounds that the infant may be affected. Pregnancy usually causes an exacerbation of the symptoms and signs which have been previously present. The initial mode of presentation in pregnancy may be as acute pyelonephritis, which is often very resistant to treatment. In the last two months of pregnancy, increasing hypertension and preeclamptic toxæmia are likely to supervene, and admission to hospital for the whole of this time seems justified. After pregnancy a remission of symptoms and signs usually occurs for a varying period. There is no evidence that pregnancy affects the ultimate prognosis of the patient, except possibly when severe pyelonephritis occurs, and there is no evidence that pregnancy stimulates the rate of growth of cyst formation within the kidney.

Occult Rupture of the Uterus.

J. M. INGRAM, R. L. ALTER AND BAYARD CARTER (*Am. J. Obst. & Gynec.*, September, 1952) define occult rupture of the uterus as rupture in which the classical symptoms of sudden pain, cessation of labour, fœtal death and maternal shock do not appear. In the Duke Hospital, a low incidence of Cæsarean section (217 cases in 16,654 deliveries, or 1·3%) has not increased the incidence of rupture of the uterus over that reported elsewhere, and the frequent pelvic delivery of patients who have had previous Cæsarean section has not resulted in one case of ruptured uterus. No vaginal delivery following Cæsarean section has been attempted unless the patient was not opposed to it, X-ray pelvimetry showed adequate measurements and architecture, the previous operator possessed adequate skill, and the previous postoperative course was afebrile; a member of the staff must be in constant attendance at the bedside during labour, and cross-matched whole blood and an operating theatre must be available. The authors state that occult rupture should be suspected in any difficult labour if descent does not occur in the presence of good labour and favourable presentation, if the patient complains of suprapubic pain and bladder tenesmus, if unexplained maternal tachycardia occurs during labour, if the fœtal heart beat ceases abruptly, or if unexplained intrapartum or postpartum hæmorrhage occurs. The maternal mortality rate of rupture of the uterus, now one of the highest encountered in any obstetrical complication, can be substantially reduced by earlier diagnosis of occult rupture.

Frank Breech and Dystocia: A New Syndrome.

R. TORPIN (*Am. J. Obst. & Gynec.*, September, 1952) presents evidence in support of a hypothesis that labour dystocia may be associated with a certain type of frank breech presentation, namely, with the sacrum directly anterior, especially in a *primigravida*. In this presentation, the heels of the fœtus lie one on each side of the maternal vertebral column, and a firm abdominal wall may hold the fœtus more or less fixed in this position. In labour, the effect is accentuated by anterior bowing of the fœtal spine, which creates a large rounded presenting part which does not engage well. The uterus, having no good outlet for its contents, tends to contract in abnormal types of labour with continuing damage to the fœtus from anoxia and danger of ultimate uterine rupture. The diagnosis is best accomplished by simple X-ray examination with a flat film of the uterus and its contents at the time of early dystocia in breech presentation.

A Psychosomatic Study of Pelvic Congestion.

C. H. DUNCAN AND H. C. TAYLOR, JUNIOR (*Am. J. Obst. & Gynec.*, July, 1952) report a psychiatrical study of 36 patients with "pelvic congestion". They define pelvic congestion as a syndrome, characterized by lower abdominal pain and parametrial tenderness, without apparent cause. There is no accepted cause of the condition, and there is no defined pathological lesion or disturbance of function. The severity of the condition may vary from a mild, passing disorder to a prolonged and incapacitating illness. The authors state that the chief clinical features are a history of dull lower abdominal pain, often worse during the premenstrual epoch and aggravated by coitus. The pain may have a superimposed cramp-like pain; it may be referred to the sacral region and may radiate to the antero-medial aspect of the thighs. Non-specific leucorrhœa, slight intermenstrual bleeding and premenstrual engorgement and pain in the breasts are associated features. Physical findings include tenderness of the parametrial tissues, cystic enlargement of one or both ovaries, diffuse enlargement of the body of the uterus, pain on stretching the utero-sacral ligaments, congestion, hypertrophy and hypersecretion of the cervix, cutaneous hyperalgesia of the lower part of the abdominal wall and nodular induration of the breasts. Alleged causes of this condition are inflammation, spastic utero-sacral ligaments and disturbed pelvic vascular function. The authors consider that alterations in local circulation and fluid distribution are probably major factors. Their study suggests that emotional factors are of importance and that pelvic congestion may be considered as one of the "stress diseases". A study of the psychological background of this series of patients and their reactions to life situations and emotions is thought to have a significant bearing on the syndrome of pelvic congestion. Ten were private patients and 26 were public patients; 32 were married and four were single, and their ages ranged from twenty-one to fifty-four years. Inquiry revealed in most cases a far from tranquil childhood and an absence of family life on which to pattern their own subsequent existence. Particularly lacking was a satisfactory relationship with a suitable mother or mother-substitute. Most of the patients were psychologically ill, but none was psychotic. Inability to function adequately as a woman, either sexually or maternally, was displayed by most. Emotional immaturity and strong dependent needs were other common traits. Two-thirds of the married women had married before the age of twenty years, often to escape from an intolerable home situation. Only six of these patients had satisfactory marital relationship. The incidence of frigidity was high among these patients, although their fertility and reproductive functions were normal. The authors studied the changes in pelvic circulation with variations in emotional state in 10 cases by noting the altered thermal conductance of the upper part of the lateral vaginal wall. They found that increased flow of blood occurred during periods of tension, particularly in relation to resentment aroused by discussion of problems with husband and children. The duration of symptoms of pelvic congestion in the patients under review varied from one to ten years. The onset of symptoms dated in most cases from pregnancy or some domestic crisis. The authors postulate that emotional stresses cause patients to react with sustained pelvic hyperæmia leading ultimately to congestion, œdema and pain.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LX.

ELECTRICAL AND DROWNING ACCIDENTS: TREATMENT.

ELECTROCUTION.

IN the past ten years about 500 persons have died in Australia from electrocution and a large number of persons have received a severe electric shock. In Victoria alone in 1952, 18 persons were electrocuted. Four of these were electrical workers who received fatal shocks during the course of their employment. Six other workmen were electrocuted, three of them because of incorrect connexions of flexible extensions, one came in contact with a mechanically damaged conduit in a roof space, and another was exposed to the hazard of live metal on a portable electric drill. The remaining eight deaths were chiefly caused by incorrectly connected flexible cords and associated accessories. One man was killed when a model aeroplane he was flying fouled high-tension wires, and a child made contact with defectively insulated aerial wires on the roof of a fernery at her home.

> In the last instance the father had left a ladder leaning against the fernery roof. Next morning the child who was playing ball in the back yard climbed the ladder to retrieve the ball when it went up on the roof. When trying to recover the ball she came in contact with the almost bare aerial wires; the insulation was old and weather worn and had largely frayed off. She received a burn on the back of the neck and a lineal burn behind the left knee.

Nearly all these deaths are preventible, and a study of the fatal cases shows that carelessness and ignorance are two of the major contributing causes.

Pathology.

When an electric current passes through the human body a number of factors influence the result. Important considerations are the type of circuit and its voltage, the nature of the contact and its duration, the resistance of the tissues, the strength of the current in the body, the path taken by the current, and the awareness of danger and the health of the individual.

While it is recognized that high voltages are dangerous, it is not so well appreciated that currents of low potential may prove fatal, especially if the contact is good and the current passes through the chest. Low voltage alternating current passing through the heart may cause ventricular fibrillation and is more dangerous than low voltage direct current. It has been stated that probably circuits of 24 volts or less may be considered as safe under practically all conditions. Here is an interesting case:

> A.L., a male worker, aged thirty-one years, was electrocuted inside a metal drum while using an electric welder. The pressure of supply in the welding circuit between the electrode and the metal structure of the drum was approximately 70 volts. The drum in which he was lying was 10 feet long and had an internal diameter of 29 inches. The deceased was welding a steel helical fin four inches deep to the internal surface of the drum. His lightly clad body was making good contact, it was a hot, humid day. The welder he was using had an open circuit secondary voltage which would be about 24 volts under working conditions. As he was manœuvering in this closely confined space, his chin apparently touched the exposed part of the electrode or the uninsulated part of the electrode holder, and he received a fatal shock. His body was extricated from the drum with difficulty. Post-mortem examination showed an area of electrical burn on the point of the chin and burns on the back of the left forearm. No other cause for death could be found; the only microscopic lesion detected was some evidence of bronchitis.

In the 18 fatal Victorian cases during 1952, 14 of the deaths were due to contact with alternating current of 230 volts. There was one instance of death from contact with direct current of 2000 volts, and two deaths were from alternating current of 132,000 volts.

Some conditions favour good contact. When a person is standing in water or on damp ground or has been perspiring freely and has a damp skin, the contact may be good, so good that in fatal cases no marks may be found on the body. Faulty radiators taken into the bathroom are a menace. One man was electrocuted with a faulty flexible connexion drooped over the bath in which he was lying.

> A youth, one very wet night, was avoiding pools of water at the side of the roadway along which he was walking, by jumping over them. When jumping near an electric light standard he lost his balance and clutched at the standard for support. He received a fatal electric shock. Post-mortem examination showed nothing more than general fluidity of the blood and a few petechiæ on the lungs. There were no marks on the body. It transpired in evidence that another person had previously received a severe shock from the same standard which was faulty.

The more prolonged the contact, in general, the more serious the effect. Contact with an electrical circuit carrying current may produce severe contraction of muscles. This muscular contraction may be so strong that the victim cannot free himself from the conductor; he is unable to let go. The current that will hold a person frozen to the conductor in this way may cause death unless the circuit is quickly broken. The muscular contraction that results when the voltage is high may be so violent as to throw the person clear of the conductor, and it may fracture bones.

When a current flows through a conductor heat is generated, and the greater the resistance, the greater the local heat produced. The resistance of the skin varies. When it is very wet the resistance of the epidermis may be only a fraction of its dry value. Saline electrodes may reduce the resistance of the skin from 5000 to 300 ohms. If the contact with the live conductor is prolonged, the resistance of the skin may be reduced by the formation of blisters. Dry skin offers the greatest resistance, and for this reason burns may be found on the body where the current enters and leaves it. The tissues at the point of contact may be subjected to great heat resulting in coagulation necrosis. The marks on the skin, commonly about the hands, vary in appearance. An electrical burn may show on the skin as a pink mark; a distinct whitish edge may be seen in the burn with surrounding erythema. There may be a single blister or a series of tiny blisters or tiny burns. The marks may look like abrasions; on the other hand, electrical burns may be very severe and very extensive. They may penetrate deeply and take a long time to heal. Charring of tissues may occur, and in severe cases fingers or portions of limbs may be lost.

The path taken by the current through the body is very important in determining the result. It is dangerous when it passes from hands to feet because the heart and lungs lie in the path and ventricular fibrillation may cause death with low voltage alternating current. The current may enter at one point on the leg and flow out the corresponding foot without danger, it may enter one leg and flow out the other without danger to important organs. If it passes through the brain or brain stem, paralysis of the medullary centres may occur and there may be great damage to the brain tissue as in judicial electrocution.

When an electric current passes through the body or portion of it, the effects vary from a slight tingling sensation to sudden death. There is often a ringing sensation in the ears and partial deafness for a time. There may be flashes of light before the eyes and brilliant luminous spots. Pain and soreness of muscles are common after the muscular contraction. Restlessness and irritability may be pronounced. Consciousness may be lost, breathing may be irregular, the pulse rapid and irregular, the skin cold and clammy, the pupils dilated. Convulsions may occur. Respiration may be suspended. The victim may recover consciousness with no after-effects. Recovery of consciousness may be followed by cerebral irritation and temporary paralysis. Sometimes there are permanent after-effects such as muscle atrophy, optic atrophy and other disturbances referable to the nervous system. There may be complicating injuries from a fall.

Death may result from respiratory centre paralysis, ventricular fibrillation, tetanus of respiratory muscles or a combination of these.

Treatment.

First Aid.

An electric shock is an emergency that calls for quick and intelligent action—prompt action to save the victim's life, and common sense and knowledge to avert a second or

even a third death. If the victim is still in contact with the conductor, if possible, the current should be switched off or the circuit broken. If this is not possible, the victim should not be grasped with the bare hands. Direct contact with the victim's skin or armpits or damp clothing should be avoided. To move him, if possible the rescuer should stand on some insulating material such as linoleum, a thick rug or a rubber mat. He should wrap dry clothing around his hands or try to push the victim away with his foot or a piece of wood or a broom. The rescuer may be able to don thick rubber gloves or stand on a rubber mat if these are at hand for an emergency such as this.

Respiratory Arrest.

If breathing has ceased, immediate artificial respiration is required (see the section on drowning for this). Artificial respiration must be maintained until recovery ensues or until the victim is dead. Cessation of respiration and failure to detect the heart beat do not necessarily mean that the victim is dead. Artificial respiration should be carried out for four hours. Maclachlan examined the reports in 700 cases of electric shock, and he found that in 479 of these people respiration had ceased, yet 323 of them were restored to life.

Ventricular Fibrillation.

With ventricular fibrillation there is no effective circulation, and death will soon follow. The conditions leading to ventricular fibrillation should be kept in mind. Cardiac massage may be effective. The passage of a 60-cycle current of one to two amperes through the heart is stated by Kouwenhoven to arrest it. "Novocain" injected directly into the heart may also be of value. Cardiac massage combined with artificial respiration may be necessary.

Further Treatment.

In addition, general treatment for shock is required as well as treatment for burns.

Prevention: Education.

The general public undertakes minor repairs to household appliances and many minor electrical tasks such as connecting flexible cords to appliances *et cetera*. This leads to many fatalities. Handyman repairs and extensions are almost impossible to control. The general public needs to be educated to appreciate the risks in handling electrical equipment. It needs instruction on how to take the necessary care with household appliances and how to prevent dangerous situations from arising. Educational films should be of value, particularly in the schools. Children are taught in school the common dangers arising from the use of electricity and how to treat shock. More intensive teaching is necessary. The introduction of a simple illustrated brochure into the household of every consumer of electricity, illustrating the dangers for the careless and ignorant person, should save many a person from an electric shock. More and more safety and more teaching are necessary to prevent the increasing number of electrical accidents.

DROWNING.

Accidental drowning accounts for many lives, and for January, 1953, in Victoria alone it was reported that there were 19 deaths from drowning, 11 of the deaths being those of children aged under fourteen years. Accidental drowning is common in the summer time from bathing and boating accidents. It also occurs at other times. Children lose their lives at any time of the year by falling into creeks, rivers, water-holes *et cetera*, and there is "death in the bath".

Pathology.

Drowning is an asphyxial process brought about by the submersion of the mouth and nostrils in a fluid medium, usually water. Complete submersion of the body is not necessary to bring about death; a child, an epileptic in a fit or a drunken person may drown in a few inches of water.

When a non-swimmer falls into water and is out of his depth, he sinks. The breath is held and his struggles together with his buoyancy bring him to the surface. He attempts to breathe at the surface and may gulp in water, throws up his arms to grasp something and sinks again. He rises and sinks and takes in more water, which on entering the air passages causes coughing, more air being expelled from the lungs. With forced breathing more water is inhaled and perhaps swallowed. In the meantime the oxygen saturation of his arterial blood is falling, and the deprivation of oxygen brings about unconsciousness. When he is unconscious with his head below the water, involuntary movements occur, there are further efforts to breathe, and more water is taken into the lungs, the remaining air in the lungs being pushed to the periphery. Ballooned distended lungs are one of the characteristics of drowning. The water which is inhaled is churned up into a fine white froth, which obstructs the air passages and prevents the entry of air into the lungs after the body is taken from the water. The length of time the man takes to drown depends among other things on his strength. Respiration ceases before heart beat in a normal subject, and in general complete submersion for five minutes will prove fatal.

A swimmer, on the other hand, swims until he is exhausted, and then may drown quickly, and the signs of drowning at autopsy may be very slight. This was illustrated in a recent ferry disaster, in which a man who had carried out many rescues and himself was a strong swimmer was drowned when people jumping off the ferry jumped on top of him. The signs of drowning at autopsy were indefinite, probably because when he was borne down by the weight of those jumping on him, he was already exhausted.

FIGURE I.

Showing the froth that obstructs the bronchial tree in drowning. Artificial respiration will fail if the bronchial tree is obstructed.

Signs of Drowning.

In recent drowning the only characteristic external evidence is the presence of a fine white froth at the nose and mouth, together with objects such as weeds, shells and sand clutched in the hands. The whitish froth may be blood-tinged; it may become evident if pressure is put upon the chest. Congestion of the eyeballs is often noted, and although the mode of death is asphyxial owing to the presence of a foreign body (water) in the air passages, the full picture of an asphyxial death is not often seen.

> In one case where it was alleged a mother deliberately drowned her child in the bath, there was marked lividity, hæmorrhages in the skin, petechiæ on the eyeballs and skin, etc., together with the characteristic findings of drowning.

Commonly in drowning, the skin is pale, the cheeks and lips may have a pinkish hue.

When the chest is opened, the fully expanded lungs may bulge from it. The lungs are heavy and may pit on pressure, and when the trachea and bronchi are opened, whitish frothy fluid is usually found filling the air passages. When the lung is cut across frothy fluid is exuded. Sand, shells, fragments of weed and material characteristic of the fluid in which drowning occurred may be found in the air passages. Water or the fluid of the medium, urine, blood *et cetera* may be present in the stomach as well as in the air passages. In general the internal signs are those of

asphyxia with dark fluid blood in the congested organs, although the lungs may not be dark in colour. Few or no petechiæ are found on the pleural surfaces or on the heart. It should be emphasized that at autopsy in some instances very little that is characteristic of drowning may be seen. Apart from those cases in which death occurs rapidly from heart arrest on the subject's going under the water or inhaling it, the pathologist meets with cases in which there is very little on post-mortem examination to show that death was due to drowning—the text-book picture is missing.

After the lapse of some days the characteristic signs of drowning pass off; then one of the most important findings is the presence of blood-stained fluid in the pleural cavities; this begins to appear in the pleural sacs about two days after death.

Examination of the blood in the right and left sides of the heart for its chloride content may be of some assistance in the diagnosis of drowning. When drowning occurs in sea-water, a much higher chloride content may be found in the blood from the left side of the heart than that from the right, and when drowning occurs in fresh water the chloride contents may be low on both sides and lower on the left side. Sara Weiden, of the Walter and Eliza Hall Institute, Melbourne, made many estimations of the chloride level in the blood on the right and left sides of the heart for us in cases of drowning. The result is of limited value. If there is a pronounced difference in the chloride level on the two sides of the heart, usually at autopsy one has little difficulty in deciding that death was due to drowning. In delayed cases in which putrefaction is evident—the type of case in which one may need some extra assistance in deciding that death was due to drowning—chloride estimations have been of little value.

Treatment.

Aim.

In a typical case of drowning with the air passages full of frothy fluid, the prime consideration should be the establishment of an adequate airway, otherwise artificial respiration may be useless. The ideal treatment would be the insertion of a bronchoscope or the passing of an endotracheal tube to suck out the contents of the bronchial tree, and then the introduction of air or oxygen directly into the lung through the tube by means of a mechanical respirator. As these means for clearing the air passages and carrying out artificial respiration are not available at the scene of the usual drowning accident, two main problems have to be faced. These are the establishment of an adequate airway under difficult circumstances, and the reestablishment as soon as possible of an adequate flow of air to and from the lungs, so that oxygen may be carried to the vital centres before they suffer irreparable damage. Various procedures are described and practised.

Clearing the Airway.

The patient should be carried from the water face downwards and with the head lower than the feet, use being made of gravity to get rid of the water in the air passages. With the head down he should be given a vigorous shake in the long axis of the body. If he is a child, he should be held up by the feet and shaken to try to empty his air passages. The patient is then placed on the ground in the prone position with the face turned to one side, on a slope head downwards if that is available or else an improvised one. If no attempt has been made to drain the air passages up to this stage, the rescuer could place his hands beneath the anterior superior iliac spines and lift the trunk off the ground to aid the exit of water. He should open the patient's mouth, pass the fingers to the back of the tongue, pulling it forward, and with the fingers sweep any foreign matter out of the pharynx and mouth. The patient may commence to breathe spontaneously at this stage; if not, artificial respiration is commenced.

Manual Artificial Respiration.

Perhaps the most widely known and practised method of performing manual artificial respiration is the prone-pressure method of Schäfer. Recent experimental work shows that other methods are more efficient. Sylvester's method and Viswanathan's method, which involve the supine position, give better expansion of the chest than Schäfer's, but suffer from the disadvantages that the tongue may fall back and the action of gravity in draining fluid away is not available; that is, they offer difficulty in maintaining the patency of the air passages.

The Holger-Nielsen method of carrying out artificial respiration has now been officially adopted by the Royal Life Saving Society and is being widely taught. This is the back-pressure arm-lift method. The patient is placed prone, with elbows bent and forearms on the ground beneath the head, and with one hand resting over the other. The cheek of the patient is placed on the uppermost hand, the face is turned slightly to one side. The operator kneels on one knee at the head of the patient and places the foot of the opposite leg near the patient's elbow. Practical experience shows that it is easier and less tiring for the operator to kneel, with both knees on the ground at the head of the patient. The operator places his hands on the patient's back in such a way that the thumbs just touch, the hands being just caudal to a line running between the arm pits. He then rocks slowly forward, elbows straight until his arms are approximately vertical, exerting steady pressure upon the chest. Then he commences to rock slowly backwards, sliding his hands to the subject's arms just above the elbows. He raises the arms until resistance and tension are felt at the patient's shoulders. Then he drops the arms. This completes a full cycle, expansion and compression phases being of equal length, the release periods being of minimum duration. The cycle is repeated twelve times per minute. This method will not be suitable if there are severe injuries to the chest and arms.

Mechanical Artificial Respiration.

If Eve's rocking apparatus is available, the patient can be transferred to this or to an improvised rocking apparatus. Eve's method relies on a piston-like movement of the diaphragm, in which the body is alternately rocked down and up in see-saw fashion on a stretcher, through an angle of 90°. Eve's apparatus consists of a stretcher and a trestle with a strong horizontal bar. The stretcher has W-shaped metal pivots fixed on the under side of the centre of the stretcher poles to prevent the stretcher from sliding and to keep the patient's abdomen off the cross-bar. The apparatus may be readily improvised, manual artificial respiration being carried out while the patient is placed on the stretcher and fixed in position. Rocking or tilting down and up is carried out with the head down at first to drain the air passages. Alternate head down and feet down rocking movements are continued at a rate of four seconds head down, three seconds feet down, each movement starting abruptly. A simple method for estimating four seconds is to repeat "1000, 2000, 3000, 4000".

Intubation.

Whenever practicable, the patient should be transferred without delay to a hospital or to medical aid where expert treatment may be given. Intubation can then be accomplished and the air passages cleared; the lungs can then be inflated directly through the airway with oxygen or air, and mechanical artificial respiration can be continued. The Victorian civil ambulances are equipped with the "R.M." resuscitator, and when the patient is handed over to the ambulance officials to be taken to hospital this resuscitator is used in place of manual artificial respiration in the ambulance. There are certain disadvantages.

Further Treatment.

During resuscitation, attention can be directed to removing wet clothing, drying the patient and restoring the heat of the body; but this should not be allowed to interfere with the carrying out of artificial respiration.

Efficient artificial respiration must be maintained until normal respiration returns or until it is certain that the patient is dead. It should be maintained for four hours. The doctor should not abandon the apparently drowned subject too soon.

> An intoxicated cook fell into a water-hole while he was assisting with some work on an electric pump on the edge of it. He swam out, and while trying to climb out grasped a faulty plug at the end of a flexible cord extension. He received an electric shock and fell back into the water. He was quickly pulled out and artificial respiration under the direction of a doctor was carried out by some medical students by Eve's method on an improvised rocker. This was continued unavailingly for four hours until the onset of rigor mortis, when there was no doubt the cook was dead.

Failure to resuscitate the apparently drowned may be due to the fact that the victim has been too long submerged, has an airway that is occluded by a foreign body, water, sand, debris *et cetera*, has died from cardiac arrest on submersion, possibly has too great a disturbance of his electrolyte balance, or has died from natural causes in the water. In a recent case resuscitation failed because the trachea and main bronchi were occluded by sand. In another instance a youth, who was a strong swimmer, was found dead at

the bottom of the local baths. Autopsy revealed an extensive subarachnoid hæmorrhage from a ruptured basal aneurysm.

After Care.

When resuscitated, the patient should remain under observation for a time, preferably in hospital. He should be watched for possible subsequent respiratory failure or pulmonary sequelæ.

Prevention.

Every child should learn to swim; his education is incomplete without it. Swimming is taught in the schools whenever practicable. Every parent should endeavour to see that his child can swim. Methods of resuscitation and what to do in an emergency must be widely known. The man who is able to swim may be able to save his own life in an emergency and the lives of others. The non-swimmer may lose his own life and cause the death of his would-be rescuer. The man who can carry out efficient manual artificial respiration may save a life, especially if the victim is one whose bronchial tree is relatively clear and has not died from myocardial failure. More emphasis needs to be placed in teaching upon the fact that the airway must be unobstructed for artificial respiration to be effective. The provision of apparatus for clearing the air passages at the life-saving centres for the use of the doctor when he arrives on the beach would seem advisable.

The teaching of this subject to the students in our medical schools must be thorough, and every doctor when he is called to treat an apparently drowned subject should realize what the problems are that confront him; in particular he should be able to pass an intratracheal tube to clear a patient's obstructed airway.

KEITH BOWDEN,
Melbourne.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Melbourne, on Wednesday, November 12, 1952.

Congenital Dislocation of the Hip.

DR. JOHN COLQUHOUN discussed problems in the management of congenital dislocation of the hip and stressed particularly the need for early diagnosis and treatment. He also described events of interest during his recent trip abroad.

Pædiatrics Overseas.

DR. RUSSELL HOWARD spoke on pædiatric impressions gained in England and America, with special emphasis on recent developments in cardio-thoracic surgery.

Why Circumcise?

DR. JOHN D. BEGG read a paper entitled "Why Circumcise?". He said that the operation of circumcision had been performed in many countries since time immemorial, but it was extraordinary how little was known of the origin and purpose of the ancient custom. It was performed in Egypt at least 4000 years B.C., probably even earlier still, and was practised as a cult among the primitive peoples of such widely separated countries as Africa and the North American continent and among Australian aborigines. It had been a Jewish custom to circumcise on the eighth day after birth since early Biblical times, and although the Koran did not specifically ordain it, all Moslems were circumcised. It was a mystery why mankind, down the ages, should have shown such a strong aversion to the retention of a useful cutaneous appendage; for, despite the enormous literature on the subject, very fully dealt with in Hastings's "Encyclopædia of Religion and Ethics", there was little real knowledge of the significance of the ancient rite.

It was tempting to ascribe a hygienic motive to the procedure, but a critical analysis of the literature did not support such a view. Even if the view was found to be empirically correct, it could scarcely have been the original motive that prompted the Egyptians to circumcise their young. Gray, in the "Encyclopædia of Religion and Ethics", concluded his summary by finding that in its inception, and late into its development, circumcision was essentially a religious custom, embracing in its broadest sense several concepts of an ideological nature. Thus, it might signify a sanctification of the reproductive organs, ensuring abundant fertility; it might be related to the ancient idea of human sacrifice—sacrifice of a small portion of the body as a propitiation of the Deity to protect the whole; or it might serve as an initiation of the youth into the full manhood of the tribe.

With primitive peoples the operation was usually carried out to the accompaniment of various ceremonials, often of a cruel and revolting nature; but in contradistinction to these, one Australian tribe performed a mock ceremony before the candidate, flourishing a large wooden knife, but leaving him untouched. That it had a deeply religious significance to the Jews was, of course, a matter of common knowledge, but whilst one maintained ancient usages, it was reasonable to examine critically a procedure which, although performed as a religious rite by one section of the community, was also carried out very frequently for supposedly surgical reasons by another.

Circumcision was performed on some 75% of male children born in Melbourne's private maternity homes, and although it was not performed routinely at the Women's or the Queen Victoria Hospital, many of the babies born in those institutions were later subjected to the operation at the Children's Hospital, or else at the pædiatric departments of the Alfred, Queen Victoria, Prince Henry's and Saint Vincent's Hospitals, as well as at the hands of private medical practitioners.

Figures revealed that in the hospitals mentioned nearly a thousand babies were circumcised annually; but, as about six thousand boys were born annually in Melbourne's two major public maternity hospitals, even if allowance was made for circumcisions performed by private practitioners, the incidence of the operation was very much lower in the public hospital class of patient than in those of the higher income groups who attended the private institutions where three-quarters of the male babies were circumcised. Gairdner, in the *British Medical Journal* of December 24, 1949, in making a comparison between children attending the best known public schools and those attending State institutions, had stated that "either the boys of well-to-do parents are suffering circumcision much too often or those of the poorer parents not often enough".

Dr. Begg then discussed the development and function of the prepuce, as described by Gairdner. He said that it developed as a ring of thickened epidermis growing forward over the glans, and during its development, which was frequently incomplete at birth, the squamous epithelium lining the inner surface of the prepuce and the outer surface of the glans was fused into one single sheet. According to Deibert, a separation into two separate linings took place by the epithelium arranging itself into whorls, in the centres of which degeneration occurred. The fusion of those degenerate areas ultimately formed a continuous subpreputial space. The process, which was seldom complete at birth, might be arrested in whole or in part at any stage of its development. Thus, again according to Gairdner, only 4% of babies had a fully retractable foreskin at birth, only 25% at six months, and 50% at the end of the first year. At the end of the third year, however, 90% of children would have a retractable foreskin; so that it was obviously fallacious to regard non-retractability of the foreskin at birth as synonymous with phimosis. Even those which could not readily be retracted at the age of three years could usually be easily rendered retractable by the separation with a probe of the residual strands of undegenerated epithelium still persisting between the inner preputial layer and the outer layer of the glans. Dr. Begg said that he had no accurate figures with which to confirm the statistics, but eleven years of observation in a provincial town where very few babies were circumcised had convinced him that true phimosis must be a very rare condition.

The function of the prepuce seemed obviously to clothe and protect the sensitive glans, not only during the early years of incontinence when the organ was in contact with urine-soaked clothes, but throughout life as well.

Like other surgical operations, circumcision carried an operative risk. The risk was admittedly small, but hæmorrhage, sepsis and anæsthetic accidents could occur, whilst, as the operation was frequently performed by practitioners of little surgical skill, mutilation of the organ was unfortunately by no means rare. The figures for Australia were not readily available, but for the five-year period from 1942 to 1947, an average of sixteen children died annually as a direct result of the operation in England and Wales. There was every reason to believe that a proportionate mortality would prevail in Australia.

Contact of the delicate tip of the glans with ammonia-soaked clothes not infrequently led to ulceration of the urethral meatus, and that, apart from being exceedingly painful, was at times very difficult to heal and could even lead to a meatal stricture. That complication of circumcision was particularly likely to occur in a child whose prepuce had become redundant from ammonia irritation—an appearance which unfortunately was regarded by some doctors as a particular indication for circumcision. Removal of the foreskin in such circumstances had been likened by a contributor to the *British Medical Journal* some years previously to "a soldier taking off his tin hat when the shrapnel was falling"; the irritation was transferred to the meatus with the inevitable result.

Dr. Begg then proceeded to examine the reasons given by parents for requesting that their child be subjected to the operation, and mentioned that inquiries from the nursing staffs of maternity hospitals revealed that the majority of babies were circumcised at the request of their parents and not on the advice of their doctors. Dr. Begg said that in his experience in questioning mothers of newborn babies on the reason for requesting circumcision, by far the commonest reason given—and this was confirmed by nursing staffs—was that the father wanted it done. The information was imparted by the mother with a slightly mysterious air as if questioning that anyone should doubt the paternal wisdom. On being further questioned, most would volunteer the information that they thought it prevented the child from wetting the bed or that it prevented in some mysterious way the development of venereal disease or the habit of masturbation. No doctor these days would seriously regard circumcision as a cure for enuresis, whilst, if the psychiatrists were to be believed, masturbation occurred in both sexes, and appeared to have no relation to the presence or absence of the prepuce. Venereologists agreed that the circumcised man who contracted syphilis or gonorrhœa was easier to manage, but there was no real evidence that the operation in any way lessened the incidence of those diseases. The matter was of still less importance at the present time, with chemotherapy and the greatly lessened incidence of venereal disease in general. Cleanliness was another reason given by parents, and it was in regard to that that a considerable misconception existed amongst both medical and non-medical people. It was true that smegma, the main source of which was degenerated epithelium lining the glans and prepuce, together with a variable amount from minute secretory glands near the frænum, did collect in the retrocoronal sulcus, but in the young child it was non-odorous and entirely harmless. Later, as mild decomposition of the smegma might occur, and as infection might then be added, it was wise to take steps to remove it. At the stage when that was likely to happen, however, separation of the two layers had taken place, and the foreskin, if not readily retractable, could usually be easily made so. Miscellaneous reasons, such as "we believe in it" and the frankly feminine one, "it looks nicer", were occasionally given.

Circumcision, therefore, though sometimes advised by the obstetrician, was more often performed by him to satisfy the parental wish. If non-retractability of the foreskin at birth was regarded with equanimity, it would be found that in time the vast majority of foreskins were so easily retracted as to fall within the scope of all but the most incapable.

It appeared to be an indisputable fact that carcinoma of the penis was virtually unknown in those circumcised before the age of five. It did, however, occur occasionally in Moslems who were circumcised between the sixth and the fourteenth years, but less commonly than in the uncircumcised. Thus Kennaway, in the *British Journal of Cancer*, comparing the incidence of the disease in Moslems with that in Hindus, among whom the operation was taboo, in fourteen hospitals in India, showed that the ratio of penile cancer to male cancer in general in Hindus was as high as 1:3·9, while that in Moslems was only 1:34·3. He suggested that failure of the operation deferred until the fourteenth year to confer the immunity produced by that performed in infancy, was due to carcinogens retained in the coronal sulcus, and if carcinoma of the penis was a common disease, there could be a very potent argument in favour of universal circumcision at an early age. The disease, however, was not a common one, particularly in western countries. An average of 6·3 cases annually were met with in Melbourne's public hospitals over a five-year period, representing only 0·3% of male carcinomata in general. Furthermore, it usually occurred in persons with a low standard of personal hygiene, and was almost invariably associated with true organic phimosis. Accordingly, a disease whose main incidence was after the sixth decade of life, and which was probably as easily prevented by simple attention to cleanliness as by circumcision, could not be cited as a convincing argument for subjecting every baby boy to the operation.

In conclusion, Dr. Begg summarized his paper by saying that circumcision, commencing as an expression of primitive man's religious emotions, was carried on largely as a response to a widespread public demand. That demand was based on a misconception of the benefits conferred by the operation, which in the great majority of children was unnecessary. As unnecessary surgery was bad surgery, the time had come when the profession should deprecate its widespread performance in infancy except for religious reasons. Indications for the operation existed only if, after the first few years, true phimosis was found to be present.

In short, the answer to the question which formed the title of the paper, "Why Circumcise?", must be in many cases "Why indeed?".

Dr. F. Stoneham said that he agreed with Dr. Begg. Phimosis could be corrected by dilatation of the foreskin with sinus forceps in most cases and circumcision was not necessary.

Dr. J. Farrer said that at birth the prepuce could be retracted in certain infants but not in others. He had been surprised at the number of infants whose prepuce was not retractable at birth, but was easily retractable two to three months later.

Dr. M. L. Powell referred to the advisability of circumcision in some cases in the tropics as a means of reducing dermatitis of the prepuce and *glans penis*. He wondered if that fact had any significance with regard to religious rites among Moslems.

Dr. Begg replied that during the last war circumcised serviceman had certainly suffered less dermatitis of the penis, and he agreed with Dr. Powell that that might have been the basis of circumcision in some religions.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

TREATMENT OF THE WOUNDED ON THE FIELD OF BATTLE.[1]

[*The Australian Medical Gazette*, August 30, 1869.]

To the Editor.

Sir,

The German medical journals have lately devoted a considerable portion of their space to the consideration of this subject. The Prussian authorities like the English found the arrangements for the treatment of the wounded insufficient. At the battle of Koniggratz numbers of valuable lives were lost from want of prompt attention. The old system was immediately altered and with this result—that at the battle of Sadown, few died from loss of blood. A series of very interesting manœuvres have lately been executed by the army. Every accident likely to occur on the field of battle was rehearsed and the surgical staff and ambulance corps were made to show that they were thoroughly conversant with their duties. Those who have been in a battle must have felt the want of efficient aid when a number of men are wounded at the same time, and from the men left to assist them not knowing how to manage slight wounds the lives of those requiring prompt attention are often lost.

It is essentially necessary that every soldier should be taught what assistance to render to a wounded comrade in the absence of the surgeon. Our authorities would do well if they made this a part of the volunteers education. There are no doubt gentlemen connected with the corps who would gladly undertake the task of delivering a few plain lectures on how fractures should be managed, bleeding from large vessels checked *et cetera*. If these colonies should be called upon to defend themselves, it will be on the volunteers—that valuable part of the community—they must rely: and it is the duty of those in authority to take

[1] From the original in the Mitchell Library, Sydney.

care that their blood is not shed unnecessarily. The Prussian system of succouring the wounded is now the most complete in the world; and any volunteer surgical staff would do well to study it carefully.

M.D.

Correspondence.

THE PERSONAL IMAGE AND UNDERLYING PHYSIOLOGICAL MECHANISMS.

SIR: We recently had the privilege of attending an inspiring lecture by the visiting neurologist, Dr. Critchley, on the above subject. Though much was admitted to be in the realms of philosophy, such vague conditions as the phantom limb, the sense of enlargement in a limb the subject of hemiplegia, and, in psychotic states, the projected *alter ego* of a de Maupassant, appeared referable to aberrations of the endogenous personal image.

As yet, there appears to be no physiological, or objective psychological frame of reference, as a substratum to the personal image. May I presume to suggest a responsible mechanism, with active motor and sensory components?

As an individual mutates from active locomotor usage to a bedridden state, and may then resume painless ambulation, the first stage in the latter operation is a shuffling gait, with little raising of the feet, while the arms hang passively at the sides. The episode of crippling may lead to undue perpetuation of this negative personal image, unless educational measures call into operation the quadrupedal element of reciprocal excitation, which is achieved by a normal swinging of the arms, and a modified attempt at goose-stepping. One such individual who had previously shuffled along, and was instructed in such a method, so invigorated her gait in her first domestic attempt that her observant cat collapsed with fright at the change in personal image, and disappeared for the day.

Returning to the serious discussion, the reality of the contralateral synthesis of motor excitation and inhibition, with an inevitable proprioceptive sensory component, as it involves diagonal limb systems, can be best appreciated from the following circumstances. It is a constituted educational exercise, in the presence of a frozen shoulder, to bend the body parallel to the ground, rest the normal hand for support on a table, advance the foot on the non-affected side forward, and swing the affected arm, in order to relax and free the shoulder-girdle musculature. It is the writer's experience that if, at the same time, the weight of the body is rhythmically swayed on to the leg, on the non-affected side, the sense of power in, and amplitude of, the swing of the shoulder, is enhanced by a reciprocal pedal excitation reflex.

More striking is the quite extraordinary impediment to normal rhythmic gait when a normal individual attempts to walk vigorously, while vigorously swinging the two arms forwards and backwards together. Persistence leads to systemic shock. Such a basic actionary mechanism, and its proprioceptive sensory element, with reflex postural cerebellar and cortical integration, must inevitably be concerned and undergo aberrations in hemiplegic states.

As pointed out by the lecturer, tumours or cerebral vascular lesions can cause a hemilateral amnesia of the body image phenomenon, and an individual so affected may wash and dry only one side of the body, trip in forgetting to lift the partly paralysed limb out of the bath, and then be satisfied to pull on only one trouser leg.

In view of the above circumstances, may not the subjective sense of enlargement of the paralysed limb be due to a psychosomatic compensation? This could be described as a proprioceptive frustration paræsthesia, in the sensory component of reciprocal excitation and inhibition.

Yours, etc.,

FRANK TRINCA.

Melbourne,
March 20, 1953.

BLOOD TRANSFUSION IN AUSTRALIA.

SIR: I feel some concern at the projected financing of the Red Cross Blood Transfusion Service in Australia by the Commonwealth Government.

Blood transfusion in Australia has been allowed to develop into a virtual monopoly. In Queensland, without the knowledge of its own Transfusion Committee, the Red Cross Transfusion Service obtained a government subsidy and for six years has been operating on that basis. Last year the State Government contributed 93·3% of the total cost of £1 12s. 8d. per donor.

So we have had experience in Queensland of nationalization of blood transfusion, and that is the reason for this letter.

Six years ago a group of us in active practice, not satisfied with the principles on which the Red Cross Transfusion Service was conducted in Queensland, established an independent blood bank which now bleeds 1200 donors yearly, has three country branches and has met every demand made upon it by any doctor who commands its services. No charge is made for the blood supplied, and the cost of production (nine shillings per donor) is only one-fifth of the average world cost of blood produced under the management of the Red Cross. We receive no government subsidy (we do not need it) and we finance the blood collection entirely from our own resources. We have not needed to make a public appeal for donors in Brisbane.

We feel that we have demonstrated over a period of six years that a blood bank can operate efficiently and economically within the framework of medical practice. We feel that the conduct of a blood bank is within the technical resources of a modern medical practice. A world-wide personal study of blood-banking practice in 1951 showed that a blood bank conducted on these lines is the most economical, efficient and flexible. This is the predominant method of blood-banking in America. The American Red Cross in its blood programme has encountered considerable resistance from the medical profession in the United States for the same reason as we have been critical in Queensland. There are defects inherent in nationalization and monopolistic control. There is a similar opposition in Canada.

The Chairman of the Red Cross National Blood Transfusion Committee in Australia (Dr. Edgar Thomson), in a report (April, 1952) to the Federal Council of the British Medical Association, states in regard to blood transfusion services in the United States of America:

> After the outbreak of war in Korea, American Red Cross again came into the transfusion field; but unfortunately met with considerable opposition, especially from certain sections of the medical profession, who were afraid that the establishment of a free Blood Transfusion Service for civilians might mean the first step in the nationalisation of the medical profession. For a considerable time there was apparently much friction.

I established the Red Cross Blood Transfusion Service in Queensland in 1940-1943, and I am concerned that events should be developing in the present direction.

I would be glad to make available further information on this viewpoint to inquirers at this critical stage.

Yours, etc.,

NOEL M. GUTTERIDGE.

"Inchcolm",
Wickham Terrace,
Brisbane.
March 21, 1953.

HEPARIN THERAPY OF SEVERE CORONARY ATHEROSCLEROSIS AND ANGINA PECTORIS.

SIR: In these days when statistics show that 31% of Australian deaths are due to cardio-vascular disease, the far greater proportion of these deaths embracing hypertension and arteriosclerosis; when we, in the course of our professional duties after the "talk", clinical, electrocardiographic and fluoroscopic examination, accord, with egregious *naïveté*, asthenic man in the fourth or fifth decade a first class life expectancy, knowing full well that he can die of a coronary occlusion almost before the ink on the proposal form is dry; for these reasons alone it would seem that the above subject transcends in importance as a field for research all other disease not excluding cancer.

It is therefore that I wish to intrude on the excellent review under "Current Comment" in THE MEDICAL JOURNAL OF AUSTRALIA of March 14 last. I trust that the author will view the following observations in the spirit intended.

Firstly, though his terms in favour of the use of heparin are guarded, they nevertheless smack, inadvertently, of course, of therapeutic blandishment and thus will encourage the irregular use of heparin by the uninitiated. For example, little will have been gained by inference unless it is

appreciated that coronary pain may be transitory and reversible, either through the development of a collateral circulation or the fact that a hæmorrhage into an atheromatous plaque may be small in extent and/or, in terms of apt if cheap parlance, what have you. The outcome will be to extend the aura and stature of the trigger-happy injectionist, who will convince himself, as well as others of his impressionable public, that he has a cure for "atherosclerosis" and "coronaries". We, in the meantime, must defer our concern for clinical research, in that it will be still in the dark as to how heparin acts on the lipoprotein molecule of the serum even to its recently discovered zeta potential factor. It is another matter to be complacent in the thought that the public purse will be invaded by some credulous and over-zealous, if not gainful, practitioner to the tune of £10 or so per patient per week while the free pharmaceutical benefits last.

I think also that it should be made clear that John Gofman and his associates would regard this aspect of heparin therapy as a small facet in their extraordinary thesis relating to the finding of a certain type of lipoprotein in the serum of atherosclerosis. Reference to their work abounds in the literature, and having seen something of Gofman and his set-up in 1950, one can readily understand why.

Somebody is sure to draw attention to Morris's statistical studies that suggest that "the incidence of coronary occlusion has increased in England over the last forty years while the overall severity of coronary atherosclerosis for the same period has declined". At first sight this might seem, if true, to invalidate Gofman's credo, but this is not necessarily so. He does not deny that there is a fatal factor in coronary artery disease that is not on an atherosclerotic basis. And might it not apply that the S_f 10-20 molecule is responsible for a particular form of atheromatous plaque, few and far between if you like, but very subject to necrosis and thus hæmorrhage from the "nutrient" *vasa vasorum*?

The answer to this latter phenomenon, the accepted major happening in coronary occlusion; also as to whether the incidence of atheroma and rupture of these tiny vessels is complementary or separable, may be resolved by obtaining serial sections of the artery when possible in every case of coronary thrombosis. Surely the importance of the subject requires that this be done. Your leader in the same issue on the National Health and Medical Research Council would appear to make the idea singularly propitious.

Yours, etc.,

149 Macquarie Street, Sydney,
March 18, 1953.

W. J. McCRISTAL.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 11, of February 26, 1953.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Appointment.—Surgeon Commander James Edward Davenport is appointed on loan from the Royal Navy, with seniority in rank of 16th April, 1945, dated 5th January, 1953.

Termination of Appointment.—The appointment of Albert Victor Morse as Surgeon Lieutenant-Commander (for short service) is terminated, dated 31st December, 1952.

Promotion.—Surgeon Lieutenant (for short service) (Acting Surgeon Lieutenant-Commander) Samuel Francis Hewitt Haughton is promoted to the rank of Surgeon Lieutenant-Commander (for short service), dated 1st February, 1953.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

To be Captains with Short Service Commissions for a period of one year.—1/8053 Alexander James Splatt, 2nd January, 1953, and 1/8054 Clifford Francis Greenslade, 23rd January, 1953.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

NX700419 Honorary Captain G. A. Cutler is appointed from the Reserve of Officers, and to be Captain (provisionally), 2nd December, 1952.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain: NX700418 G. P. Dodd, 9th December, 1952, NX700417 J. H. Steele-Smith, 19th December, 1952, and NX700419 G. A. Cutler, 16th December, 1952.

Citizen Military Forces.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—The following officers are appointed from the Reserve of Officers, and to be Captains (provisionally): Honorary Captains 2/130107 C. B. Saunders, 29th October, 1952, and F2/1075 C. D. Chalmers, 10th December, 1952.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—To be Captain (provisionally), 9th January, 1953: 3/101022 Alexander Glynn Mathew.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—To be Honorary Captains: Guiseppe Borzi, 19th January, 1953, and Charles Steele Haughton, Geoffrey Arthur Hocker and Geoffrey Byford Ilett, 22nd January, 1953.

5th Military District.—To be Honorary Captain, 4th January, 1953: William Ewan Sandover. Honorary Captain W. E. Sandover is retired, 13th January, 1953.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The undermentioned officers are granted acting rank as indicated, 2nd October, 1952: (Wing Commander) Squadron Leaders G. A. Leyland (04397), C. W. Fitton (033030); (Squadron Leader) Flight Lieutenant L. A. Watson (034058).

Air Force Reserve: Medical Branch.

The following former officers are appointed to commissions with the rank of Flight Lieutenant: H. A. Copeman (016442), 7th May, 1952, J. G. H. Refshauge (409447), 21st October, 1952, J. W. Gardiner (257561), 23rd October, 1952, A. R. Shepherd (443328), 3rd November, 1952.

The following are appointed to commissions with the rank of Flight Lieutenant: Hubert Desmond O'Brien (257895), 6th November, 1952, Winfield Robert Curtis Bennett (257892), 20th November, 1952.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course for the Diploma in Clinical Pathology.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that, provided a sufficient number of candidates is offering, a course for the diploma in clinical pathology will begin on July 20, 1953, for a period of seven months. The course of study is divided into three groups: group I, pathological anatomy and hæmatology; group II, bacteriology and parasitology; group III, biochemistry. The closing date for applications is June 22, 1953, and further details of the diploma and course may be obtained from the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483.

Week-End Course at Wagga Wagga.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at the Wagga Wagga Base Hospital, in conjunction with the Southern Districts Medical Association, on Saturday and Sunday, May 16 and 17, 1953. The programme will be as follows:

Saturday, May 16: 2 p.m., registration; 2.30 p.m., "Pulmonary Tuberculosis: Planning Treatment", Dr. W. C. B. Harvey; 4 p.m., "Recent Advances in Anæsthesia: Especially Relaxants and Positive Pressure Anæsthesia", Dr. A. Distin Morgan.

Sunday, May 17: 10 a.m., "Bronchiectasis in Children", Dr. D. G. Hamilton; 11.30 a.m., "The Significance of Hæmoptysis", Dr. W. C. B. Harvey; 2 p.m., "Control of Respiration during Anæsthesia", Dr. A. Distin Morgan; 3.30 p.m., "Dietary Disorders in the Second Year (Cœliac Disease *et cetera*)", Dr. D. G. Hamilton.

The fee for attendance is £3 3s. Those wishing to attend are requested to notify Dr. John Oliver, Honorary Secretary, Southern Districts Medical Association, 69 Baylis Street, Wagga Wagga, telephone number 2568, as soon as possible.

Obituary.

QUINTO ERCOLE.

THE following appreciation of the late Dr. Quinto Ercole is prepared from information supplied by Dr. J. W. Quilter.

Quinto Ercole died at "Campewarra", Grenfell, on February 2, 1953. He was born at Campli, Abruzzi, Central Italy, in 1870, one of a family of four children. His parents died while he was still a child, and his early life was difficult. He studied medicine at the University of Bologna, Italy, and graduated with honours in 1896, receiving the degrees of Doctor of Medicine and Doctor of Surgery. After three years' work in his profession he went to Australia and practised in Elizabeth Street, Sydney, until 1902. In that year he married Adele Veron, daughter of an old French family, and for the next two years practised in White Cliffs, New South Wales. Then he returned to Sydney in 1904, took out naturalization papers and practised in William Street, Sydney, until 1906, when he commenced practice in Grenfell. During the first World War, Ercole served overseas with the Australian Imperial Force, but was discharged from that force as medically unfit in 1917. However, he continued to serve as medical officer on army troopships as long as he was able to do so, and then returned to resume his practice in Grenfell. In 1934 he retired from practice and purchased the "Campewarra" property, where he lived until his death.

Quinto Ercole played a prominent part in the life of Grenfell. He made a notable contribution to the development of the local district hospital. For many years prior to his retirement he filled the post of government medical officer. From 1928 to 1934 he was an alderman and deputy mayor in the Grenfell Municipal Council. He played an important part in bringing about the installation of electricity, water and sewerage systems in the municipality. He contributed much to the development of the Grenfell Literary Institute. He was vice-president of the Returned Soldiers' League. In recognition of his work for the community, the Council paid him the rare honour of placing his portrait in the Council Chambers. It can indeed be said that his services were always readily available in any movement intended to advance the interests of the town and district. He is survived by his widow, one son and one daughter.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additional qualifications have been registered: Duval, Ferdinand (M.B., B.S., 1937, Univ. Sydney, D.M.R. (London), 1945), F.F.R. (London), 1952; Sussman, Ewen (M.B., B.S., 1946, Univ. Sydney), M.R.C.O.G., 1952.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners: Shaw, Charles Gordon, M.B., B.S., 1951 (Univ. Melbourne); Meredith, John Evan, M.B., B.S., 1952 (Univ. Sydney); Jagger, Justin Ross, M.B., B.S., 1952

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MARCH 21, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	4(2)	..	..	..	..	..	..	4
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	3	..	3
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	2	..	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	1	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1(1)	4(1)	10(9)	..	..	..	4	..	19
Diphtheria	27(13)	10(8)	5(3)	..	4(1)	..	..	..	46
Dysentery (Bacillary)	..	..	..	5(5)	4(4)	..	..	..	9
Encephalitis	..	1	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	2	..	..	..	..	..	..	2
Infective Hepatitis	..	9(7)	..	..	11(10)	..	1	..	21
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1	5(2)	1	..	..	..	..	..	7
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	16(9)	13(2)	19(3)	14(10)	..	1	..	..	63
Puerperal Fever	..	..	2	..	..	..	..	..	2
Rubella	..	25(8)	..	..	2(1)	..	..	..	27
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	16(9)	66(31)	2(1)	2(2)	4(4)	1	..	2	93
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	23(15)	16(1)	7(2)	10(6)	7(4)	4(2)	..	..	67
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1]Figures in parentheses are those for the metropolitan area.

(Univ. Melbourne); Hunn, John McLeod, M.B., B.S., 1951 (Univ. Melbourne); Auerbach, Egon, M.B., B.S., 1952 (Univ. Sydney); Houston, John Lyster, M.B., B.Ch., B.A.O., 1951 (Queen's University, Belfast).

The following additional qualifications have been registered: Connolly, Hugh Glynn, M.F.A., R.A.C.S., 1952; Hart, Daniel Roberts Lloyd, F.R.C.S. (England) (Ophth.), 1952; Muller, Edmund, M.F.A., R.A.C.S., 1953.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners: Christie, Marjorie R., M.B., B.S., 1951 (Univ. Sydney); Holmes, Alan Boyd, M.B., B.S., 1950 (Univ. Sydney); Anderson, Duncan McKenzie, M.B., Ch.B., 1939 (Univ. Belfast); Wade, Eric Arnold, M.B., B.S., 1952 (Univ. Sydney).

Deaths.

THE following death has been announced:

HALL.—Edwin Cuthbert Hall, on April 9, 1953, at Sydney.

Medical Appointments.

Dr. H. B. Taylor and Dr. C. B. Cox have been appointed analysts within the meaning and for the purposes of the *Pure Food Act*, 1908, in the Department of Public Health of New South Wales.

Dr. H. G. Wallace, Dr. C. J. Cummins, Dr. F. W. Fraser, Dr. H. B. Taylor and Dr. H. W. T. Chenhall have been appointed members of the Advisory Committee for the purposes of the *Pure Food Act*, 1908, in the Department of Public Health of New South Wales.

Dr. C. J. Cummins has been appointed a member of the Board of Health of New South Wales.

Congresses.

INTERNATIONAL CONGRESS OF RADIOLOGY.

THE seventh International Congress of Radiology is to be held in Copenhagen from July 19 to 25, 1953. His Majesty King Frederik IX is patron and the Honorary President is His Excellency Professor Niels Bohr. The President and Secretary-General is Professor P. Flemming Moller. Unfortunately the whole of the accommodation in Copenhagen is booked out for the period of the Congress and further enrolments will be accepted only from those who have accommodation already arranged or who have friends with whom they can stay.

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Handley, Donald Alfred, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 43 South Esplanade, Glenelg, South Australia.

Riley, John, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 6 Pier Street, Glenelg, South Australia.

Harris, James Dunbar, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), "The Glen", Crafers, South Australia.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Brown, Ross Alvarez, M.B., B.S., 1950 (Univ. Adelaide); Stobie, Peter James, M.B., B.S., 1952 (Univ. Adelaide); Ashton, Thomas James, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Torr, Thomas Harold, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Kalinovsky, Peter Victor, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Kalinovsky, Galina Vera, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Basheer, Keith Cameel, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Browne, Noel John, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Murray, Donald Bruce Seymour, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Law-Smith, David Gordon, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Sando, Maurice James Wilson, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952).

Diary for the Month.

APRIL 28.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 30.—New South Wales Branch, B.M.A.: Branch Meeting.
APRIL 30.—South Australian Branch, B.M.A.: Listerian Oration.
MAY 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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